REVIEW ARTICLE

Pursed Lips Breathing

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Introduction

Pursed-lips breathing (PLB) is a maneuver that is frequently taught to patients with chronic obstructive pulmonary disease (COPD) in respiratory physiotherapy programs to improve breathing efficiency and better manage dyspnea during activities of daily living. Researchers first became interested in PLB when emphysema patients were clinically observed to breathe instinctively with the lips semi-closed in an attempt to minimize dyspnea. Although this technique had been described and recommended in the mid-1950s and beginning of the 1960s, the first studies designed to establish the benefits and physiological effects of PLB were not published until the mid-1960s. Even now—forty years later—there are few studies on PLB in the literature and the factors underlying its efficacy are not well understood. While most studies have focused on patients with COPD, some have found that PLB may be beneficial in certain neuromuscular diseases and exercise-induced asthma. In this paper, we review the published studies (Table) in which PLB was evaluated individually or compared to other techniques.[1-15] For clarity, we have divided this review into separate sections in which the effects of PLB on lung function and arterial gases, breathing pattern, and respiratory muscles are discussed. Finally, the clinical effects of PLB are reviewed.

Effect of PLB on Lung Function and Arterial Gases

Nerini et al[13] and Bianchi et al[15] studied changes in chest wall lung volumes in COPD patients performing PLB. The authors observed that patients showed a significant decrease in end-expiratory lung volume (EELV), with greater reduction occurring with more severe obstruction—as defined by forced expiratory volume in 1 second (FEV_1). In addition, they noted that these patients normally performed PLB instinctively. Ugalde et al[12] and Spahija et al[10] also found similar results with respect to EELV. Mechanically, EELV represents the point of equilibrium between the forces of elastic recoil of the lungs and the chest wall. A decrease in EELV represents an increase in the elastic recoil of the chest and potentially more energy for inspiration, which may occur passively as a result of the potential energy of the chest wall at the end of expiration.[16-20]

Mueller et al[3] evaluated the effect of PLB on PaO_2, $PaCO_2$ and oxygen saturation (SaO_2) in COPD patients at rest and during exercise. At rest, they found a significant increase in PaO_2 and SaO_2 and a significant decrease in $PaCO_2$; the results were the same for all patients, whether or not they perceived benefits from the PLB. No significant changes in arterial gases during exercise were observed. Tiep et al,[4] using an ear oximeter to study the effect of PLB on SaO_2, found a significant increase. Ugalde et al[12] found similar results in both healthy subjects and patients with myotonic muscular dystrophy (MMD). However, these results were not confirmed by Roa et al,[6] who found a minimal increase in SaO_2 that was not statistically significant. These findings suggest that PLB may improve gas exchange at rest, but not during exercise.

Effect of PLB on Breathing Pattern

The breathing pattern describes the process of air exchange between the environment and the lungs. The variables used to reflect breathing pattern are sensitive to any changes in the frequency and/or volume of air exchanged during respiration. In addition, the breathing pattern allows us to study the mechanics and regulation of ventilation in the context of the many factors that affect oxygen supply and demand.

The efficacy of PLP in regulating respiration in COPD patients at rest—by significantly decreasing breathing frequency and increasing tidal volume—has been

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Manuscript received 4 November, 2003. Accepted for publication 11 November, 2003.

TABLE
Summary of Clinical Trials Reviewed*

Authors	Year	Type of Clinical Trial	Sample	Variables Studied
Schmidt et al[1]	1964	Control group	10 healthy controls, 10 emphysema, and 7 asthmatics	Vital capacity
Thoman et al[2]	1966	No control group	21 COPD	V_T, f, $PaCO_2$, lung volumes
Mueller et al[3]	1970	Randomized	12 COPD	SaO_2, V_T, f, PaO_2 and $PaCO_2$, VO_2, and VCO_2
Tiep et al[4]	1986	Randomized crossover	12 COPD	SaO_2, V_T, f, V_E
Wardlaw et al[5]	1987	Randomized with control group	10 asthmatics	FEV_1
Roa et al[6]	1991	No control group	12 COPD	VO_2, V_E, V_T, f, SaO_2, Pg, Ppl, Pdi, and dyspnea
Breslin[7]	1992	Self control	13 COPD	SaO_2, f, V_T, Ppl, Pg, Pdi, and TTdi
Spahija et al[8]	1993	No control group	6 COPD	V_T, T_E, T_I, T_{TOT}, Pes, dyspnea, and VAS
Breslin et al[9]	1996	Self control	13 COPD	EMG of abdominal muscles, Pga, V_T, f, T_I/T_{TOT}, and Borg scale
Spahija and Grassino[10]	1996	Self control	11 healthy subjects	Breathing pattern, EELV, respiratory mechanics, and muscle recruitment
Van der Sahans et al[11]	1997	Self control	12 asthmatics	FVC, V_T and EMG of the scalene, parasternal, and abdominal muscles
Ugalde et al[12]	2000	Control group	13 healthy, 11 MMD	EMG of abdominal muscles, abdominal, chest wall, plethysmography, V_T, f, SaO_2, Borg, and EELV
Nerini et al[13]	2001	No control group	5 COPD	Chest wall volumes, V_T, f, V_E, and EELV
Jones et al[14]	2003	Randomized	30 COPD	VO_2, f
Bianchi et al[15]	2003	No control group	30 COPD	Chest wall volumes, EELV

*COPD indicates chronic obstructive pulmonary disease; MMD, myotonic muscular dystrophy; V_T, tidal volume; f, breathing frequency; SaO_2, arterial oxygen saturation; VO_2, oxygen consumption; VCO_2, carbon dioxide output; V_E, minute ventilation; FVC, forced vital capacity; FEV_1, forced expiratory volume in 1 second; Pg, gastric pressure; Ppl, pleural pressure; Pdi, transdiaphragmatic pressure; Pes, esophageal pressure; TTdi, diaphragmatic tension-time index; T_E, expiratory time; T_I, inspiratory time; T_{TOT}, total time of respiratory cycle; VAS, visual analog scale; EMG, electromyography; EELV, end-expiratory lung volume.

described by authors such as Thoman et al,[2] Muller et al,[3] Tiep et al,[4] Roa et al,[6] and others.[7-9,19] Thus, this breathing pattern seems to be more effective than spontaneous breathing in COPD patients. Mueller et al[3] identified 2 types of patients. The first type—who reported symptom relief—had an increase in tidal volume and a significant decrease in breathing frequency when using PLB; the second type—those reporting no improvement—also showed a significant decrease in breathing frequency, although no change in tidal volume. These findings support the idea that not all patients benefit from this breathing pattern. On the other hand, Ugalde et al,[12] studying the effects of PLB on MMD patients, found the effects to be similar to those reported for COPD patients. The authors attributed breathing pattern improvement to a decrease in EELV. Spahija and Grassino,[10] likewise, noted that a decrease of 3% to 4% in EELV in COPD patients could be interpreted as biomechanically advantageous during inspiration. The same phenomenon has been reported by other authors.[20-23] Finally, van der Schans et al,[11] using a graduated (5 cm H_2O) PEEP valve, confirmed the benefits of experimentally-induced PLB in asthmatic patients. Their findings showed an increase in tidal volume both when no obstruction was present and during propranolol-induced bronchospasm attack. While very little is known about the efficacy of PLB during exercise, some authors have found that PLB has similar effects on COPD patients and healthy subjects with respect to tidal volume and breathing frequency at rest.[3,10] These 2 studies suggest that, when comparing the breathing pattern during exercise with and without PLB, the pattern promoted by PLB is closer to respiration in basal conditions. Some authors, such as Mueller et al,[3] observed that PLB caused a significant decrease in minute ventilation output in COPD patients, both at rest and during exercise. However, other studies have found no significant differences in minute ventilation.[4,6,13] In a recent study of MMD patients and healthy subjects, Ugalde et al,[12] observed improvement similar to that described by Mueller et al[3] in both groups. Although findings related to minute ventilation are few and controversial, we can state that the increase in tidal volume during PLB is sufficient to maintain minute ventilation unchanged, despite the decrease in breathing frequency.

Some authors have studied the time variables of the breathing pattern during PLB. Breslin[7] and Spahija et al[8] found a significant reduction in the respiratory duty cycle (ratio of inspiratory time to total time) and in the diaphragmatic tension-time index in COPD patients. Saphija and Grassino,[10] in a study of healthy individuals, observed that PLB increased expiratory time and total time both at rest and during exercise; in addition, inspiratory time increased significantly during exertion.

In short, the impact of PLB on the breathing pattern seems to be positive, both at rest and during exercise,

because it promotes prolonged expiration with a decrease in EELV, leading to lower breathing frequency and higher tidal volume; the end result is an improvement in ventilatory efficiency.

Effect of PLB on Respiratory Muscles

The respiratory muscles are responsible for maintaining adequate ventilation. The force and resistance of respiratory muscles can be assessed by measuring several variables, such as the maximum respiratory pressure (both inspiratory and expiratory), maximum voluntary ventilation, and transdiaphragmatic pressure. The dynamic measurement of the respiratory muscles during the respiratory cycle is accomplished primarily by studying intrathoracic pressure (pleural pressure) measured at the esophagus and abdominal pressure measured in the gastric area or, alternatively, by plethysmography of the chest surface. Respiratory muscle function can also be evaluated by noninvasive (surface electrodes) or invasive electromyography of the chest wall muscles.

Roa et al,[6] who studied the work of breathing and ventilatory muscle recruitment during PLB in COPD patients, observed a significant decrease in gastric and pleural pressures during inspiration and an increase in respiratory work. This increase was attributed to an increase in the work of the chest wall (intercostal) muscles as a result of decreased work of the diaphragm, caused by a more negative pleural pressure and a decrease in gastric pressure during inspiration. Breslin,[7] in addition to confirming the aforementioned results, noted abdominal muscle recruitment during the entire respiratory cycle and a significant decrease in the diaphragmatic tension-time index. In another study, Breslin et al[9] measured gastric pressure and performed surface electromyography in COPD patients, confirming an increase in gastric pressure during expiration and in contractions of all the abdominal muscles studied. Spahija and Grassino[10] and Ugalde et al,[12] studying healthy subjects and MMD patients, respectively, reached the same conclusions. Given these findings, we can conclude that abdominal muscle recruitment and chest wall expansion at rest and during exercise is greater with PLB. On the other hand, when van der Schans et al[11] studied the effects of PLB on the tonic and phasic electromyographic activity of the scalene, parasternal, and abdominal muscles of asthma patients after the administration of propanolol, they observed that the increased work of these muscles occurred principally during phasic activity.

In short, PLB can be said to cause a change in the pattern of respiratory muscle recruitment, increasing recruitment of the accessory muscles of the chest wall and increasing abdominal muscle activity throughout the entire respiratory cycle while, at the same time, decreasing diaphragmatic muscle recruitment. All these changes lead COPD patients to breathe more efficiently and consume less oxygen; as a result, the propensity of the diaphragm to become fatigued during crises or physical exercise decreases.[14]

Clinical Effects of PLB

Schmidt et al[1] were the first authors to hypothesize about the impact of PLB on dyspnea in patients with emphysema, who perform PLB instinctively. Their findings showed that decreased breathlessness in these patients was produced by the reduction in the variability of expiratory flows, causing a decrease in the Bernoulli effect created by airflow and thereby reducing the tendency of the airways to collapse. Nonetheless, Breslin et al,[9] using the Borg scale to study dyspnea in COPD patients, compared spontaneous breathing to PLB breathing and found that PLB—despite increasing ventilation—did not reduce the degree of dyspnea and even significantly increased it in some patients. These findings were confirmed by Roa et al[6] with COPD patients and Ugalde et al[12] with MMD patients and healthy subjects. Ugalde et al[12] were also able to show that PLB increased fatigue and respiratory effort, as measured by the Borg scale. Finally, Spahija et al[8] assessed the effects of PLB on COPD patients during submaximal exercise. None of the patients had dyspnea at baseline; however, during exercise, dyspnea was more severe in patients who performed PLB than in those who did not. From these studies, we can conclude that the effect of PLB on dyspnea in COPD and MMD patients is still unclear because results published to date are not consistent with the relief of breathlessness reported by some patients.

Wardlaw et al[5] observed that most patients experienced no bronchoconstriction (as measured by FEV_1) when performing PLB during hyperventilation-induced bronchoconstriction. This observation led them to suggest that PLB may benefit patients with exercise-induced asthma.

Jones et al[14] studied oxygen consumption and the clinical implications of having COPD patients perform breathing exercises (including PLB). Compared to spontaneous breathing, oxygen consumption was significantly reduced in all of the breathing patterns studied: diaphragmatic breathing (DB), PLB, and a combination of DB and PLB. PLB resulted in the lowest oxygen consumption, followed by DB and then the combination of PLB and DB. Based on their results, the authors suggested that COPD patients be taught to use breathing patterns that consume less oxygen in order to minimize the metabolic demand of respiration.

Conclusion

Given the results reported in the literature reviewed, we conclude that PLB can improve breathing function in patients with primary or secondary respiratory diseases. The breathing pattern associated with PLB produces a more physiological and efficient ventilation. The expiratory resistance provided by the lips leads to significant changes in the temporal variables of the breathing pattern and in respiratory muscle recruitment. As a result, tidal volume increases, gas exchange

improves, and oxygen consumption decreases. No evidence has been put forth, however, to support an impact of PLB on breathlessness. In some patients, the sensation of breathlessness seems to decrease with PLB, but when dyspnea is measured, it increases in many others. The increased dyspnea during PLB may be caused by the greater involvement of the accessory muscles of respiration, which are less resistant to fatigue. Despite the discrepancies among the limited number of studies on the effects of PLB, we believe that the maneuver should be included in respiratory physiotherapy programs to improve breathing efficiency in patients with COPD, asthma, and neuromuscular diseases with respiratory involvement, such as MMD.

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