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Influence of pursed-lip breathing on heart rate variability and cardiorespiratory parameters in subjects with chronic obstructive pulmonary disease (COPD)

Influência da respiração freno-labial sobre a variabilidade da frequência cardíaca e parâmetros cardiorrespiratórios em pacientes com doença pulmonar obstrutiva crônica (DPOC)

Ramos EMC¹, Vanderlei LCM¹, Ramos D¹, Teixeira LM¹, Pitta F², Veloso M³

Abstract

Objectives: To assess the effects of pursed-lip breathing (PLB) at rest on the behavior of heart rate (HR) and its variability, and on variations in blood pressure (BP), respiratory rate (RR) and pulse oxygen saturation (SpO₂) in subjects with chronic obstructive pulmonary disease (COPD). **Methods:** Sixteen subjects with COPD (seven in GOLD stage I, three in GOLD stage II and six in GOLD stage III; mean age 64±11 years; mean FEV₁ 60±25% of predicted value) were assessed at rest, in a seated position, under the following conditions: ten minutes of normal breathing without PLB (R1), eight minutes with PLB (R2) and ten minutes of normal breathing once more (R3). HR was recorded, beat-to-beat, by means of a Polar S810 heart monitor. The RMSSD index (root mean square of the difference between successive R-R intervals) was determined. BP, RR and SpO₂ were also assessed during the trials. ANOVA for repeated measures followed by the Tukey test and Kruskal-Wallis test were used for data analysis, with a 5% significance level. **Results:** There was a significant increase in the RMSSD index during R2, in comparison with R1. The HR variation between inspiration and expiration was 8.98 bpm, and the variation between HR at rest and HR with PLB was 8.25 bpm. During R2, RR decreased and SpO₂ increased significantly in comparison with R1 and R3. BP values did not show significant changes. **Conclusions:** The results showed that PLB produced significant changes in HR, RR and SpO₂, and did not alter BP in subjects with COPD. Furthermore, analysis of the RMSSD index showed that PLB promoted increased parasympathetic activity in these subjects, thus indicating that this technique influenced the autonomic cardiac modulation.

Key words: Chronic obstructive pulmonary disease; pursed-lip breathing; heart rate variability.

Resumo

Objetivos: Avaliar os efeitos da respiração freno-labial (RFL), em repouso, sobre o comportamento da frequência cardíaca (FC) e sua variabilidade e variações na pressão arterial (PA), frequência respiratória (FR) e saturação parcial de oxigênio (SpO₂) em pacientes com doença pulmonar obstrutiva crônica (DPOC). **Métodos:** Dezesesseis pacientes com DPOC (7: estágio GOLD I, 3: estágio GOLD II e 6: estágio GOLD III; média de idade=64±11; média de VEF₁=60±25% do predito) foram avaliados em repouso, na posição sentada, nas seguintes condições: 10 minutos respirando normalmente sem RFL (R1), 8 minutos com RFL (R2) e 10 minutos respirando novamente normalmente (R3). A FC foi registrada, batimento a batimento, por meio do frequencímetro Polar S810, e o índice RMSSD (raiz quadrada da média das diferenças entre intervalos RR sucessivos) foi determinado. PA, FR e SpO₂ foram também avaliados durante o protocolo. ANOVA para medidas repetidas, seguida pelo teste de Tukey e teste de Kruskal-Wallis foram usados para análise dos dados, com nível de significância de 5%. **Resultados:** Ocorreu aumento significativo no índice RMSSD durante R2 em comparação com R1. A variação na FC inspiração/expiração foi de 8,98 bpm, e a variação na FC em repouso/RFL foi de 8,25 bpm. Durante R2, FR diminuiu e SpO₂ aumentou significativamente em comparação a R1 e R3. Os valores de PA não apresentaram modificações significativas. **Conclusões:** Os resultados mostraram que a RFL produziu modificações significativas na FC, FR e SpO₂ e não alterou a PA em pacientes com DPOC. Além disso, a análise do índice RMSSD mostrou que a RFL promoveu aumento da atividade parassimpática nesses pacientes, indicando que essa técnica influencia a modulação autonômica cardíaca.

Palavras-chave: doença pulmonar obstrutiva crônica; respiração freno-labial; variabilidade da frequência cardíaca.

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Introduction ::::

Pursed-lip breathing (PLB) is frequently used by patients with chronic obstructive pulmonary disease (COPD) in pulmonary rehabilitation programs and in their activities of daily living, in order to improve breathing efficiency and to decrease dyspnea during these activities¹⁻⁵. When the lips are closed during PLB, resistance is imposed on the expiratory flow, thus leading to a positive expiratory pressure in the airways. This stabilizes and dislocates the point of equal pressure in the bronchial tree from the periphery to a more proximal location. This, in turn, decreases the resistance of the airways to the release of air and consequently decreases the residual volume⁶⁻⁸. In addition, expiratory delay promotes homogenous emptying of the lungs, thereby maintaining the intrabronchial pressure and favoring gas exchange and ventilation. This promotes increased pulse oxygen saturation (SpO_2) and partial pressure of oxygen in the blood (PaO_2), and decreased partial pressure of carbon dioxide in the blood (PaCO_2)^{1-4,6}. There is also an increase in tidal volume⁹, greater recruitment of expiratory muscles and a reduction in respiratory rate (RR), with a decrease in minute ventilation (VE) at rest^{1,2}.

Despite the therapeutic importance of PLB, some of the effects of this technique have not been well established, such as its influence on the cardiovascular system. Respiratory rate and rhythm not only influence the respiratory system but also exert a direct influence on the cardiovascular system. Some respiratory maneuvers may lead to an overload of this system, thereby activating arterial baroreceptors, chemoreceptors and cardiopulmonary receptors that are linked to the central nervous system. This leads to responses caused by the activation of the autonomic nervous system, which regulates heart rate (HR) and blood pressure (BP)¹⁰⁻¹⁴. Therefore, in addition to respiratory responses, PLB may also influence cardiovascular responses, although there is still a lack of studies investigating this topic.

The objective of the present study was to assess the effects of PLB at rest on the behavior of HR and its variability, and on BP, RR and SpO_2 , in subjects with COPD.

Methods ::::

Sixteen subjects with COPD were selected and assessed in this study. The protocol was submitted to and approved by the Research Ethics Committee of the School of Science and Technology, Presidente Prudente Campus, Universidade Estadual Paulista (FCT/UNESP) (case no. 212/2007). The subjects were informed about all of the experimental procedures that would be used and about the objectives of the study. Upon agreement,

they signed a consent form. The criteria for inclusion were: 1) diagnosis in accordance with international guidelines¹⁵; and 2) no medication ingestion and no associated diseases that might interfere with autonomic cardiac control.

Protocol

Spirometry testing was performed using the Spirobank spirometer (MIR, Rome, Italy). The test was performed in accordance with international standards¹⁶. The interpretation of the spirometric data and disease staging were performed in accordance with the Global Initiative for Obstructive Lung Disease (GOLD)¹⁵.

The subjects were asked not to have any alcoholic and/or stimulating drinks, such as coffee or tea, less than 12 hours before performing the trial. In order to avoid possible interference with the study variables caused by medications, the subjects who were using maintenance drugs (bronchodilators, mucolytics, anti-inflammatory agents and other drugs) were also asked to interrupt their use over the 12-hour period preceding the trial.

The experimental trial was performed at a temperature of 21 to 24°C and relative humidity of 50 to 60%. The subjects sat down on a chair and remained at rest, breathing spontaneously for ten minutes (R1). After that, they were asked to perform PLB for eight continuous minutes (R2) and then they remained at rest for another ten minutes, breathing spontaneously (R3). During PLB, the subjects were asked to relax the neck and shoulder muscles and to breathe in slowly through the nose for approximately two seconds, within the tidal volume range, keeping the mouth shut. Expiration was slow and prolonged with pursed lips⁸. The same researcher collected all the data and instructed the subjects concerning the protocol and how to perform the PLB technique.

Cardiorespiratory monitoring was performed by means of assessing RR, SpO_2 , BP and HR. RR was obtained by counting the number of cycles completed in one minute, as assessed by chest movements. SpO_2 measurements were performed using a BCI 3303 oximeter (BCI International, Waukesha, Wisconsin, USA). BP was measured using a Tycos aneroid sphygmomanometer (Welch Allyn Tycos 705014, USA) and a stethoscope (Littmann Classic II SE, 3M Health Care, USA), placed on the subject's left arm. HR was recorded beat-to-beat throughout the experimental trial using a Polar S810 heart monitor (Polar Electro, Kempele, Finland). This device had previously been validated for this procedure and for heart rate variability (HRV) analysis^{17,18}.

HRV analysis over the periods before, during and after PLB was performed by taking a five-minute interval within each of these periods, covering the portion of the period when the subjects presented greatest signal stability. Only series containing

more than 256 RR intervals were used in the analysis¹⁹. In the temporal series, automatic filtering was performed using the Polar Precision Performance SW software (version 4.01.029), complemented by manual filtering, in order to eliminate artifacts or cardiac arrhythmias that could interfere in the HRV analysis^{20,21}. The temporal series was filtered before selecting periods of longer stability.

HRV analysis was performed in the time and frequency domains using the following indices: 1) time domain: in this domain, the RMSSD index from the R-R intervals in milliseconds was used, which corresponds to the root mean square of the difference between successive RR intervals^{13,22}; 2) frequency domain: in this domain, we used low-frequency power in normalized units (LFnu, 0.04-0.15 Hz), high-frequency power in normalized units (HFnu, 0.15-0.4 Hz) and LF/HF (ratio of absolute LF power to absolute HF power). The spectral analysis was calculated using the Fast Fourier Transform algorithm. The HRV Analysis software²³ was used to calculate these indexes.

HR was monitored throughout the trial, and RR, SpO₂, BP were recorded during the last minutes of each trial period.

Table 1. Anthropometric characteristics and disease staging of the 16 COPD subjects studied.

Age (years)	64±11
Weight (kg)	66±14
Height (cm)	164±6
BMI (kg/m ²)	24±4
FEV ₁ (l)	1.53±0.60
FEV ₁ (% of predicted value)	60±25
GOLD stage (I / II / III)	7 / 3 / 6

BMI=body mass index; FEV₁=forced expiratory volume in the first second; GOLD=Global Initiative for Obstructive Lung Disease.

Statistical analysis

ANOVA for repeated measurements, followed by the Tukey test, was used to examine differences in RMSSD, RR, SpO₂ and BP during the three protocol periods: at rest with spontaneous breathing (R1), using PLB (R2) and after using the technique (R3). The Kruskal-Wallis test was used to assess whether the study variables presented differences according to the degree of disease severity. Statistical significance was set at $p < 0.05$.

The study power was calculated using the GraphPad StatMate software, version 2.00 for Windows (GraphPad Software, San Diego, California, USA). It took into consideration the rest phases and PLB and showed that, for all the variables, with the current sample size and 5% as the significance level (two-sided test), the power of the study for detecting differences between the variables was greater than 80%.

Results

Data from 16 subjects were analyzed (12 men and four women). The subjects' general characteristics and their classification according to the degree of airflow obstruction are shown in Table 1. Subjects with different degrees of airflow obstruction, reflecting different COPD stages according to the GOLD classification, showed no significant differences in RR, SpO₂, systolic and diastolic BP and RMSSD index at any assessment time during the protocol (R1, R2 and R3). Therefore, the different degrees of airflow obstruction were not taken into consideration for data analysis.

HR varied during PLB, increasing during inspiration and decreasing during expiration (Figure 1). The variation in HR

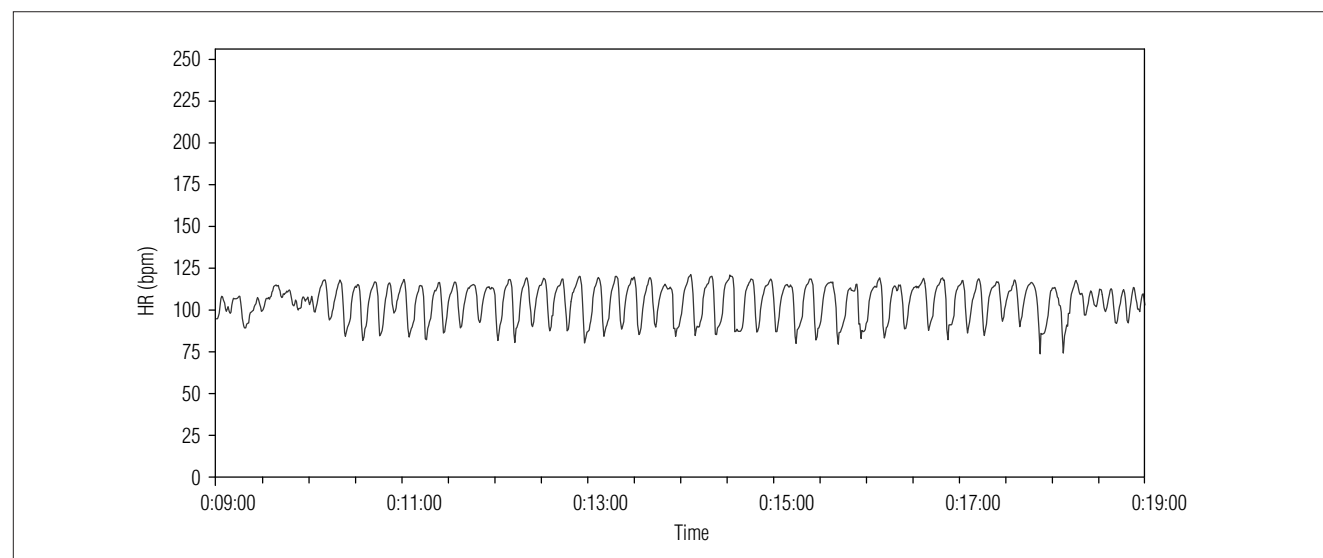


Figure 1. Heart rate pattern during one minute of rest, eight minutes of pursed-lip breathing and one minute of recovery.

between inspiration and expiration, calculated based on the difference between the mean peak HR during inspiration and the mean minimum HR value during expiration^{24,25}, was 8.98 bpm. The mean peak HR during inspiration was 83±14 bpm and the mean expiration peak was 74±10 bpm. The variation in HR between resting and PLB was 8.25 bpm and was calculated based on the difference between the mean peak HR during inspiration while performing PLB and the mean HR value during the minute at rest before PLB²². The mean peak HR during inspiration while performing PLB was 83±14 bpm and the mean HR at rest was 75±12 bpm.

With regard to the RMSSD index (Table 2), the analysis showed significant differences between R1 and R2 ($p<0.05$). No significant difference was observed between R3 and R1 or between R3 and R2 ($p>0.05$). There were no significant differences in the LFnu and HFnu indices, or in the LF/HF ratio, between the times studied (Table 2; $p>0.05$ for all data).

Table 3 shows the systolic and diastolic BP, RR and SpO₂ values during the trial. The analysis did not show significant differences between systolic and diastolic BP during R1, R2 and R3. For RR and SpO₂, the analysis showed significant differences between R1 and R2 ($p<0.05$) and between R2 and R3 ($p<0.05$). No significant differences were observed between R1 and R3 for RR and SpO₂ ($p>0.05$).

Discussion

PLB is a technique used by many COPD patients in order to reduce dyspnea while exercising, performing activities of daily living or even at rest. Our results showed that PLB induced HR changes that were similar to what is observed in respiratory sinus arrhythmia. This is a cardiorespiratory phenomenon characterized in mammals by HR or R-R interval fluctuations that are in phase with inhalation and exhalation²². It has been associated with better efficiency in pulmonary gas exchanges, thereby producing better alveolar perfusion and ventilation^{25,26}. During the breathing cycle, there is an increase in HR during the inspiratory phase, followed by a decrease during the expiratory phase^{25,26}. This occurs because calm and serene inspiration leads to parasympathetic inhibition, thus increasing HR as muscle sympathetic activity is suppressed. In contrast, during calm and serene expiration, there is a decrease in HR due to parasympathetic activation, together with facilitation of muscle sympathetic activity²².

Respiratory sinus arrhythmia is frequently used to assess vagal tonus, because it increases and decreases the parasympathetic influence when the autonomic nervous system is appropriately balanced. According to Holnloser²⁷ and Taylor²⁸, inspiration/expiration variations greater than 9 bpm show that

Table 2. Heart rate variability indices at rest with spontaneous breathing (R1), using PLB (R2) and after using the technique (R3).

	Rest (R1)	Pursed-lip breathing (R2)	Recovery (R3)
RMSSD	16.94±3.47	22.89±4.41*	19.58±3.35
LFnu	72.53±3.42	59.68±7.30	71.35±2.79
HFnu	27.47±3.42	40.33±7.30	28.65±2.79
LF/HF	3.67±0.63	3.64±0.93	3.14±0.47

Data are shown as mean ± standard error of mean; * $p<0.05$ versus R1; RMSSD=root mean square successive difference between the RR intervals; LFnu=low-frequency power in normalized units; HFnu=high-frequency power in normalized units; LF/HF=LF/HF ratio.

Table 3. Systolic and diastolic blood pressure (BP), pulse oxygen saturation (SpO₂) and respiratory rate (RR) at rest with spontaneous breathing (R1), using PLB (R2) and after using the technique (R3).

	Rest (R1)	Pursed-lip breathing (R2)	Recovery (R3)
Systolic BP	124±22	117±21	126±21
Diastolic BP	74±10	76±11	74±10
SpO ₂	96±2	98±1*	96±3
RR	19±4.6	10±3*	18±5

Data are shown as mean ± standard deviation. * $p<0.05$ versus R1 and R3.

vagal functioning is intact. The values observed in the present study were very close to 9 bpm: the variation in HR between inspiration and expiration was 8.98 bpm and the variation in HR between resting and inspiration during PLB was 8.25 bpm. The HR inspiration/expiration variation was within the normal range reported by Gilbert²⁹ (from 8 to 14 bpm).

The RMSSD index from the R-R intervals was analyzed and showed statistically higher values during PLB, compared with the values obtained at rest during spontaneous breathing. This suggests that PLB promotes increased vagal modulation, which may be related to decreased RR, thereby intensifying the respiratory sinus arrhythmia²². Although it did not reach statistical difference, the RMSSD index after PLB (R3) remained higher than the initial resting values (R1), thus suggesting that increased parasympathetic cardiac activity and decreased sympathetic activity were present during the recovery phase after PLB. According to Paschoal et al.³⁰, the increased parasympathetic activity during the recovery phase of the dynamic physical activity occurs to protect the heart against arrhythmias.

With regard to the variability indices obtained in the frequency domain, no significant differences were observed between the times analyzed. Nevertheless, the rise observed in the HFnu index, modulated exclusively by parasympathetic activity^{31,32}, and the consequent reduction in the LFnu index, modulated by sympathetic and parasympathetic activity^{31,32}, also suggest that PLB promoted a rise in the vagal modulation through the application of this technique.

Systolic and diastolic BP values were not significantly altered. However, there was a tendency towards decreased systolic BP and a slight increase in diastolic BP during PLB. These

variations may be related to oscillations in intrathoracic pressure produced by respiratory movements, which determine increases and decreases in systolic debit, thereby contributing towards the changes in BP levels that occur during the respiratory cycle¹².

Previous authors have suggested that PLB improves SpO₂ and decreases RR¹⁻⁴. Both findings were reproduced in the present study. According to the abovementioned authors, the decrease in RR during PLB is possibly related to better control over the respiratory cycles and increased duration of expiration, thus leading to a higher tidal volume. The increase in SpO₂ during PLB suggests that the increased duration of expiration (and consequent increase in tidal volume) leads to homogeneous lung emptying, thereby maintaining the intrabronchial pressure and favoring both gas exchange and ventilation^{1-4,6}. Moreover, it should also be mentioned that patients who experience reductions in dyspnea while using PLB might also have reductions in end-expiratory lung volume and greater reserves in inspiratory muscle pressure-generating capacity. This

would lead to increased end-inspiratory lung volume, which is somewhat independent of tidal volume⁴.

Although Camillo et al.³³ showed that COPD severity was not significantly related to HRV indices, the limited number of subjects classified as GOLD stage II in the current sample might be considered a limitation of the present study. In addition, the inclusion of a healthy control group (without COPD) may elucidate whether the results shown for the COPD group were effectively caused by the PLB maneuver or whether they were partially affected by the disease itself.

Conclusions

In summary, the present results showed that PLB led to changes in HR, RR and SpO₂, and did not alter BP significantly. Furthermore, analysis of the RMSSD index showed that PLB promoted increased parasympathetic activity, thus indicating that this technique influenced autonomic cardiac modulation.

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