Effect of Slow Diaphragmatic Breathing Technique on Heart Rate, Blood Pressure and Peripheral Oxygen Saturation in Hypertensive Elderly

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ABSTRACT

Slow breathing can be used as a complement for hypertension treatment. The aim of the study was to verify the effect of a breathing technique on cardiac autonomic function in elderly (N = 22, 76.36 ± 7.93 years old, 13 women); thirteen had optimal to Normal High Blood Pressure (ONH group) and nine had Mild to Moderate Isolated Systolic Pressure (MMS group). In both groups, vital signs were collected in two breathing conditions: i) Baseline (B)- participants breath at their normal pace; ii) Diaphragmatic (D)- participants breath at a slow pace, predominantly abdominal. Considering all participants, results revealed that during D condition Respiratory Frequency (RF) and Heart Rate Frequency (HR) were significantly reduced, and Peripheral Oxygen Saturation (SpO2) significantly augmented. In MMS group, both groups significantly augmented SpO2. Additionally, in B condition, MMS group had significantly less SpO2 than ONH group; but, in D condition no significant difference was found between groups. Similarly, in B condition, MMS group had significantly higher Diastolic Pressure (DP) and Pulse (P- the difference between SP and DP). Consequently, during the D breathing, MMS subjects benefited of better blood pressure and peripheral oxygenation conditions, with probable enhancement of Heart Rate Variability (HRV). Results of this study support the hypothesis that an easily learned diaphragmatic breathing technique, inexpensive and non-intrusive, can help elderly with isolated systolic hypertension, to remediate its effects on vital signs, as a non-clinical complementary treatment.

KEYWORDS: Breathing technique; Elderly; Blood pressure; Peripheral oxygen saturation

INTRODUCTION

A slower respiration usually involves the use of the diaphragm, probably without increase of respiratory workload. Slow breathing increases baroreflex sensitivity [1-32,14-22], which is lower in essential hypertensive than in normotensive subjects [28,29]. Because the baroreflex modulates cardiac vagal and sympathetic outflow to the sinus node in the heart [33-36], slowing breathing frequency results in a reduction of Heart Rate (HR) [37] and reduces muscle nerve sympathetic activity [2-16]. That’s why it has been suggested that reducing breathing frequency can be used...
as a complement for hypertension treatment [26,38], Probably, an increase of Oxygen Saturation (SpO₂) may also occur, as in patients with chronic heart failure, regardless of the amount of ventilation [27] and particularly for cases with lower SpO₂ values [4].

Heart Rate Variability (HRV) represents the variations of the RR intervals, i.e., the time elapsing between two consecutive R waves and has been used as an expression of physiological factors that modulate the heart rhythm [6]. The Sympathetic Nervous System (SNS) acts to increase heart rate and the Parasympathetic Nervous System (PNS) acts to lower heart rate [35]. Hypertension results in a decrease in HRV, and an increase in sympathetic activity, detected by a greater low frequency power (LF) and a smaller high frequency power (HF) at supine rest; evolving with passive tilting to some increase and decrease, respectively; meaning a cardiac sympathetic tone increase and a cardiac vagal tone and modulation decrease [17], with negative consequences on blood pressure, particularly in severe hypertension [25].

Therefore, the purpose of the present study was to verify the effect of a diaphragmatic breathing technique on cardiac autonomic function in hypertensive elderly, by analysis of HR, BP, SpO₂ and short-term HRV parameters.

METHODS
Sample
The sample was composed of 22 elderlies (76.05 ± 7.98 years of age), 13 women, 13 with optimal to normal high blood pressure (prehypertension), and 9 with mild to moderate systolic isolated pressure [8-24]. Based on clinical history, ambulatory and successive experimental blood pressure registrations, no signs of white coat effect or masked hypertension were detected. One participant was taking medication for depression, and four signs of white coat effect or masked hypertension were detected. and successive experimental blood pressure registrations, no isolated pressure [8-24]. Based on clinical history, ambulatory and successive experimental blood pressure registrations, no signs of white coat effect or masked hypertension were detected. One participant was taking medication for depression, and four signs of white coat effect or masked hypertension were detected. Informed consent was obtained.

Procedures and Data Treatment
Respiratory Cycles per Minute (RF) were recorded through direct observation of thoracic and/or abdominal movements. Interval RR was carried out through Polar V800 [15] Pic Classic Check sphygmomanometer was used to collect Systolic (SBP) and Diastolic (DBP) blood pressure every minute; and, Comed Eco Oximeter was used to collect Peripheral Oxygen Concentration (SpO₂), every 15 seconds. Records were conducted at the participants' places, with subjects in the supine position, in a quiet environment, with room temperature between 19 and 22 °C. Participants were instructed not to smoke, drink alcohol or coffee for 4h before data collection [21-23].

The experimental session was structured according to the following sequence: 6min of rest with normal breathing, taken as Baseline (B), and 6mn Diaphragmatic Breathing Technique (D).

In a previous training session, of about 20mn, participants were instructed about and practiced the D, as follows: (1) put one hand on your chest and the other on your belly, (2) breath only through your nose, (3) fill your belly with air; and then let it go out slowly. No pace of breathing was imposed and no control the depth of breathing was made, so that each person could maintain comfortable breathing pace [4,11,38], The recording of B was always done first.

For HRV analysis gHRV software was used [33]. HR data were automatically filtered, employing adaptive thresholds in order to reject in correct beats [34], whose values exceeded the cumulative mean threshold, and to eliminate points outside acceptable physiological values. A linear interpolation method, with filtered non-equispaced HR signal, was used to obtain frequency domain analysis [39]. For the spectral analysis a 4Hz signal interpolation was used: with a window size of 120s and a time shift of 60s.

Data were statistically treated with the program IBM-SPSS, version 24. Shapiro-Wilk test was used to verify data normal distribution. Wilcoxon test (W) was used for within group comparison, with Monte Carlo test. Effect size (r) and Wilcoxon rank-biserial correlation coefficient (rrb) were calculated. Mann-Whitney U test (Z) was used for between group comparison, with Monte Carlo test. Effect size (r) and Mann-Whitney Glass rank-biserial correlation (rrb) were calculated [12]. Spearman's Rank Order Correlation (rs) was used for determining association among variables.

RESULTS
Overall, during D, and compared to B, participants significantly reduced RF, HR, SP, DP and P, and significantly augmented SpO₂ (Table 1).

Table 1: Descriptive statistics (Mean ± Standard Deviation, Median), Wilcoxon test (W), effect size (r), and Wilcoxon rank-biseral correlation coefficient (rrb), of collected vital signs (below) for the sample (N= 22), in baseline (B) and diaphragmatic breathing (D) conditions.

<table>
<thead>
<tr>
<th>Vital Sign</th>
<th>B</th>
<th>D</th>
<th>GL</th>
<th>W</th>
<th>p</th>
<th>r</th>
<th>rrb</th>
</tr>
</thead>
<tbody>
<tr>
<td>RF</td>
<td>18.95±3.46, 19</td>
<td>13.38±3.23, 12</td>
<td>132</td>
<td>9.582</td>
<td>0.0001</td>
<td>0.83</td>
<td>0.98</td>
</tr>
<tr>
<td>HR</td>
<td>72.25±9.42, 71</td>
<td>70.73±9.64, 71</td>
<td>6632</td>
<td>21.513</td>
<td>0.0001</td>
<td>0.26</td>
<td>0.25</td>
</tr>
<tr>
<td>SpO₂</td>
<td>94.27±2.42, 95</td>
<td>95.84±2.08, 96</td>
<td>528</td>
<td>13.332</td>
<td>0.0001</td>
<td>0.58</td>
<td>0.56</td>
</tr>
<tr>
<td>SP</td>
<td>136.43±22.22, 136</td>
<td>123.57±18.04, 124</td>
<td>132</td>
<td>6.14</td>
<td>0.0001</td>
<td>0.53</td>
<td>0.46</td>
</tr>
<tr>
<td>DP</td>
<td>72.86±8.32, 72</td>
<td>68.81±8.13, 68</td>
<td>132</td>
<td>5.215</td>
<td>0.0001</td>
<td>0.45</td>
<td>0.39</td>
</tr>
<tr>
<td>P</td>
<td>63.58±18.34, 65</td>
<td>57.76±16.28, 57</td>
<td>132</td>
<td>5.033</td>
<td>0.0001</td>
<td>0.44</td>
<td>0.39</td>
</tr>
</tbody>
</table>

RF: Respiratory Frequency; HR: Heart Rate Frequency; SpO₂: Peripheral Oxygen Saturation; SP: Systolic Pressure; DP: Diastolic Pressure; P: Pulse.
If we compare the participants by their blood pressure status, both groups maintain significantly less RF, HR, SP, P, and significantly more SpO_2 in D condition than in B condition. Additionally, MMS group significantly reduced DP (Table 2). Between groups, no significant differences were found in RF, in B condition (Z(132) = .489, ns, r = .04, rrb = .09) and in D condition (Z(132) = .797, ns, r = .07, rrb = .05), and a very lowly correlated and powered significant difference for HR in B condition (Z(6847) = 5.745, p < .0001, r = .06, rrb = .09) and in D condition (Z(6847) = 5.856, p < .0001, r = .07, rrb = .09). However, for SpO_2 in B condition, ONH group revealed significantly higher values than MMS (Z(528) = 6.645, p < .0001, r = .29, rrb = .34); however, that difference disappeared in D condition (Z(528) = 1.468, ns, r = .06, rrb = .07). For SP, ONH group preserved significantly better values in B and D conditions (Z(132) = 9.569, p < .0001, r = .83, rrb = .98; Z(132) = 6.149, p < .0001, r = .54, rrb = .63, respectively), and in P (Z(132) = 8.638, p < .0001, r = .75, rrb = .89; Z(132) = 6.150, p < .0001, r = .54, rrb = .63, respectively); but in DP, MMS group evolved from significant higher value in B condition to non-significant difference in D condition (Z(132) = 6.255, p < .0001, r = .54, rrb = .64; Z(132) = .646, ns, r = .06, rrb = .07, respectively); (Table 2).

Table 2: Descriptive statistics (Mean ± Standard Deviation, Median), Wilcoxon test (W), effect size (r), and Wilcoxon rank-biserial correlation coefficient (rrb), of collected vital signs (below) for the sample (N= 13), in baseline (B) and diaphragmatic breathing (D) conditions.

<table>
<thead>
<tr>
<th>Vital Sign</th>
<th>Group</th>
<th>B</th>
<th>D</th>
<th>n</th>
<th>Z</th>
<th>p</th>
<th>r</th>
<th>rrb</th>
</tr>
</thead>
<tbody>
<tr>
<td>RF</td>
<td>ONH</td>
<td>18.77±3.79, 19</td>
<td>13.62±3.20, 13</td>
<td>78</td>
<td>7.492</td>
<td>0.0001</td>
<td>0.85</td>
<td>1</td>
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<tr>
<td></td>
<td>MMS</td>
<td>19.22±2.93, 19</td>
<td>13.04±3.26, 12</td>
<td>54</td>
<td>6.016</td>
<td>0.0001</td>
<td>0.82</td>
<td>0.96</td>
</tr>
<tr>
<td>HR</td>
<td>ONH</td>
<td>72.56±8.77, 72</td>
<td>71.20±8.11, 71</td>
<td>4338</td>
<td>17.848</td>
<td>0.0001</td>
<td>0.27</td>
<td>0.27</td>
</tr>
<tr>
<td></td>
<td>MMS</td>
<td>71.68±10.50, 69</td>
<td>69.87±11.94, 71</td>
<td>2324</td>
<td>12.126</td>
<td>0.0001</td>
<td>0.25</td>
<td>0.22</td>
</tr>
<tr>
<td>SpO_2</td>
<td>ONH</td>
<td>94.90±1.90, 95</td>
<td>96.05±1.76, 96</td>
<td>312</td>
<td>10.036</td>
<td>0.0001</td>
<td>0.57</td>
<td>0.55</td>
</tr>
<tr>
<td></td>
<td>MMS</td>
<td>93.35±2.78, 94</td>
<td>95.54±2.44, 96</td>
<td>216</td>
<td>8.951</td>
<td>0.0001</td>
<td>0.61</td>
<td>0.57</td>
</tr>
<tr>
<td>SP</td>
<td>ONH</td>
<td>122.01±12.98, 124</td>
<td>118.5±10.53, 120</td>
<td>78</td>
<td>2.949</td>
<td>0.001</td>
<td>0.34</td>
<td>0.28</td>
</tr>
<tr>
<td></td>
<td>MMS</td>
<td>157.26±15.11, 154,5</td>
<td>138.17±20.28, 134</td>
<td>54</td>
<td>5.400</td>
<td>0.0001</td>
<td>0.73</td>
<td>0.7</td>
</tr>
<tr>
<td>DP</td>
<td>ONH</td>
<td>69.00±6.20, 69</td>
<td>68.81±8.13, 68</td>
<td>78</td>
<td>1.294</td>
<td>ns</td>
<td>0.15</td>
<td>0.13</td>
</tr>
<tr>
<td></td>
<td>MMS</td>
<td>78.43±7.86, 76</td>
<td>69.57±9.22, 70</td>
<td>54</td>
<td>5.377</td>
<td>0.0001</td>
<td>0.73</td>
<td>0.74</td>
</tr>
<tr>
<td>P</td>
<td>ONH</td>
<td>53.01±14.22, 58,5</td>
<td>50.26±11.44, 53,5</td>
<td>78</td>
<td>2.769</td>
<td>0.01</td>
<td>0.31</td>
<td>0.31</td>
</tr>
<tr>
<td></td>
<td>MMS</td>
<td>78.83±11.69, 76</td>
<td>68.59±16.20, 64,5</td>
<td>54</td>
<td>4.313</td>
<td>0.0001</td>
<td>0.59</td>
<td>0.5</td>
</tr>
</tbody>
</table>

RF: Respiratory Frequency; HR: Heart Rate Frequency; SpO_2: Peripheral Oxygen Saturation; SP: Systolic Pressure; DP: Diastolic Pressure; P: pulse by blood pressure status (Mild to Moderate Isolated Systolic Pressure- MMS, n= 7; and, Optimal to Normal High blood pressure subjects- ONH, n= 13).

Considering all participants, in the B condition, RF isn’t associated with SpO_2 (rs(132) = -.051, ns), however, in the D condition a significantly inverse association is observed (rs(132) = -.342, p < .0001), meaning that when longer and predominantly diaphragmatic breathing was requested, less breathing cycles per minute became associated with greater peripheral oxygen saturation and vice-versa. However, when we split participants by blood pressure status, additional information is available; ONH group revealed significant inverse association in B and D conditions (rs(78) = -.302, p < .01; rs(78) = -.301, p < .01, respectively); whereas MMS group revealed a divergent pattern, in B condition a significant direct association is present (rs(54) = .390, p < .01), meaning that greater breathing frequency is associated with greater peripheral oxygen saturation, whereas in the D condition a significant inverse association appeared (rs(54) = -.406, p < .01), meaning that these participants clearly reverse the association among RF and SpO_2. If we remind that both groups had significantly more SpO_2 in the D condition than in the B condition (Table 2), we can suppose that for MMS participants a greater RF in B condition is a solution that their body resorts to ensure enough SpO_2, naturally with a supplementary effort for their hearts, which can be advantageously substituted by the D technic, with a significant reduction of HR (Table 2). An additional proof of this abrupt physiological change in the MMS group is the absence of association of RF among conditions (rs(54) = .09), and in D condition (Z(132) = 6.255, p < .0001, r = .54, rrb = .64; Z(132) = .646, ns, r = .06, rrb = .07, respectively); (Table 2).

Table 3: Descriptive statistics (Mean ± Standard Deviation, Median), Wilcoxon test (W), effect size (r), and Wilcoxon rank-biserial correlation coefficient (rrb), for heart rate variability parameters for the sample (N= 22), in baseline (B) and diaphragmatic breathing (D) conditions.

<table>
<thead>
<tr>
<th>HRV Parameters</th>
<th>B</th>
<th>D</th>
<th>GL</th>
<th>W</th>
<th>p</th>
<th>r</th>
<th>rrb</th>
</tr>
</thead>
<tbody>
<tr>
<td>HRVi</td>
<td>7.44±3.35, 6.76</td>
<td>8.07±3.69, 7.17</td>
<td>22</td>
<td>1.964</td>
<td>0.05</td>
<td>0.42</td>
<td>0.36</td>
</tr>
<tr>
<td>HF</td>
<td>107.00±195.40, 47.60</td>
<td>189.52±285.88, 57.91</td>
<td>22</td>
<td>2.549</td>
<td>0.01</td>
<td>0.54</td>
<td>0.44</td>
</tr>
<tr>
<td>LF</td>
<td>144.59±198.07, 61.46</td>
<td>194.36±242.80, 99.35</td>
<td>22</td>
<td>1.964</td>
<td>0.05</td>
<td>0.42</td>
<td>0.44</td>
</tr>
</tbody>
</table>

HRVi: Heart Rate Variability Index; HF: High Frequency
ACKNOWLEDGEMENT

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REFERENCES


