Hyperbaric oxygen improves nasal air flow

P. VERA-CRUZ¹, J. CROCA¹ and C. ZAGALO²

¹Serviço de Otorrinolaringologia, Hospital da Marinha, Lisboa, Portugal
²Instituto Superior de Ciências da Saúde Egas Moniz, Monte da Caparica, Portugal.

Objective: We investigated whether hyperbaric oxygen (HBO₂) treatment is able to cause any changes in the nasal peak inspiratory flow (NPIF) values of patients submitted to this therapy.

Study design: NPIF was measured in a group of 13 patients who were submitted to at least 10 sessions of 75 minutes long HBO₂ treatments over a period of 20 days. HBO₂ was prescribed to the patients to treat hearing loss, diabetic ulcers or chronic inflammatory disease. Three timings were chosen to perform the NPIF measurements: during HBO₂, five minutes before and five minutes after the treatment.

Methods: For NPIF evaluation, the highest inspiratory flow of three inspirations was recorded. To search for statistical differences between NPIF measurements at the three different timings of the HBO₂ treatment, we have analysed the data using the repeated measures ANOVA test with the Epsilon lower bound correction for the F ratio.

Results: NPIF values were significantly higher when the patients were inside the HBO₂ chamber when compared with NPIF measurements obtained in the same individuals five minutes before starting or five minutes after ending the treatment. A small but significant increase in NPIF values was detected in patients five minutes after stopping the HBO₂ treatment, in comparison with values obtained five minutes before initiating the therapy. NPIF values remained stable along the 10 HBO₂ sessions, i.e. with repetition of the HBO₂ treatments, NPIF values were not further enhanced.

Conclusions: Exposure to HBO₂ causes significant improvement in nasal air flow. This increase is restricted mostly to the period that the patients are inside the hyperbaric chamber. Further investigations are needed to determine the relative contributions of enhancement in air pressure and in oxygen concentration (that characterize HBO₂) in the enhancement of nasal air flow. The herein finding may be helpful in future investigations on the treatment of nasal or sinus diseases.

INTRODUCTION

Nasal mucociliary function is known to increase both in hyperbaric and in pure oxygen breathing (1). Reversely, under hypobaric hypoxia conditions — for instance, when breathing at high altitude — there is decrease in nasal flow (2). The controlled environmental conditions inside a hypobaric chamber have refuted the hypothesis that nasal blockage at altitude is due to the inhalation of cold and dry air (3). It is pertinent to recall that normal nasal function is of great importance to prevent dive accidents involving the middle ear and the nasal sinuses (4).

Hyperbaric oxygen therapy (HBO₂) consists in the delivery of 100% oxygen to patients at pressures that are two to three times higher than those at sea level (1). The patients are kept inside a hyperbaric chamber, and the increased pressure aims at enhancing the amount of oxygen dissolved in the plasma. The therapeutic actions of HBO₂ are related with direct physical effects of oxygen on blood and tissues and also with a number of secondary physiological and biochemical benefits (5,6). The Undersea and Hyperbaric Medical Society has approved the use of HBO₂ in the treatment of several clinical conditions that are ameliorated by increase in the availability of oxygen to tissues.

Since it has been generally considered that hypobaric hypoxia decreases nasal flow, we have decided to test the hypothesis that HBO₂ may have the opposite effect, i.e., that this treatment, contrary to general expectations, will improve nasal air flow.
For that, we have evaluated nasal function before, during and after HBO$_2$ treatment of patients by determining the nasal peak inspiratory flow (NPIF) values.

The question is relevant for patients with nasal obstruction related with rhinitis, when these patients are submitted to HBO$_2$ with the goal of treating pathologies that are not located in the airways. If oral breathing, which is common in patients with nasal obstruction, will be replaced by physiological nose breathing, then the HBO$_2$ session can be more comfortable and, in addition, middle ear and the sinus symptoms are expected also to decrease.

Finally, it is pertinent to recall that the subjective sensation of nasal obstruction modulates the values for nasal air flow (7), such as the NPIF that we have measured in the herein investigation.

**MATERIALS AND METHODS**

**Hyperbaric oxygen (HBO$_2$)**

The HBO$_2$ treatment took place in a multipurpose hyperbaric chamber (Haux® – Starmed 2200) in the presence of a nurse. All HBO$_2$-treated patients finished 10 sessions of HBO$_2$ therapy at 2.5 ATA (1 atmosphere absolute – ATA) for 75 minutes per session and over a total period of 20 days.

They underwent one session each day and at the same hour. The total length of patient stay in the chamber for each session was 100 minutes because of the time needed for compression (10 minutes) and decompressing (15 minutes). The pressure was obtained by compressed air, and the patients breathed 100% humidified oxygen through tightly fitted (nose and mouth) masks, expiring through large tubes connected with valves to the space outside the chamber.

**Patients**

A group of 13 patients were chosen for this study. HBO$_2$ had been prescribed to these patients because of the diagnosis of sudden neurosensorial hearing loss (eight patients), lower-extremity diabetic ulcers (three patients) radiation-induced cystitis (one patient) and Buerger’s disease (one patient). The group was made up of eight men and five women, with ages ranging 21-76 years, with a mean of 51.9±18.3 years.

This research project was approved by the Ethics Committee of the Portuguese Navy Hospital (Lisbon), where the investigation was done. All subjects gave their written informed consent to participate in the study. Exclusion criteria to eliminate patients from the study were the following:

- all criteria that exclude patients from HBO$_2$ therapy;
- upper-airway anatomical abnormalities;
- history of asthma, rhinitis, upper airway infection (shorter than six weeks);
- previous trauma or nasal surgery;
- drug addiction;
- cigarette smoking or professional exposure to air pollutants.

In order to investigate whether the NPIF values of the patients were different from those of a healthy population, we also obtained spirometric data from a second group of individuals. This second group of healthy volunteers was made up of four men and eight women. They presented an age range of 25-57 years with a means of 43.5±11.7. Their body mass indexes (BMIs) were not significantly different from those of the group of patients. Two NPIF measurements, separated by 100 minutes, were performed at normal oxygen and pressure conditions in this healthy population.

**Study design**

This study took place over 20 days in the Portuguese Navy Hospital, Lisbon, Portugal. The hospital building is located 20 meters (65.62 feet) above sea level. The temperature inside the hyperbaric chamber was 21ºC (69.80ºF). During the treatment, temperature decreased 0.5ºC (32.90ºF) under maximum pressure. The relative humidity in the air inside the chamber was 55%. All NPIF measurements were made using an In-check™ ATS97 inspiratory flow meter (Clement Clarke International, UK) fitted with a nose and mouth mask. The patients placed the spirometer against their faces, ensuring a close seal, and then inhaled, as strongly as they were able to through the nose.
from residual volume, with a closed mouth. The highest inspiratory flow of three inspirations was recorded.

The HBO2-treated group performed an NPIF evaluation five minutes before beginning the treatment, a second one when they reached the maximum pressure and a last one five minutes after the end of the treatment. The numerical data were compared using repeated measures ANOVA applied with the Epsilon lower bound correction for the F ratio. The SPSS™ 16.0 Version statistical software package was used. Statistical significance was considered at p<0.05.

RESULTS

We obtained values of the NPIF parameter before, during and after HBO2 treatment of a group of 13 patients. In order to compare NPIF values from these three timings, we have chosen repeated measures ANOVA as the statistical method, since multiple comparisons were sought at three different timings.

The assumption of sphericity for repeat measures ANOVA, checked by using the Mauchly test, was violated. Therefore, a multivariate test was applied and showed that the average NPIF of the subjects differs significantly between the times of measurement (p = 0.001). In order to compare the mean values of NPIF at different times, post-hoc tests were applied.

We found that HBO2 treatment of patients (Table 1, page 152) caused a transient NPIF increase from 149,4 ± 12,0 l/min (at sea level and before treatment) to 176,4 ± 16,0 l/min (under increased pressure, 2.5 ATA, and enhanced oxygen concentration). This 18% increase was statistically significant (p = 0.001). After the HBO2 treatment the NPIF of the patients was 158,4 ± 13 l/min; this value was 6% higher than the value before treatment and was significant different from it (p = 0,007). The difference between NPIF values during and after treatment is also statistically significant (p = 0,009)

During the HBO2 treatment, NPIF had a linear rise of 3 l/min/day in all evaluations (before HBO2, at 2.5 ATA and after HBO2). Linear modeling is quite significant: p = 1,8 ± 10-5 for the evaluation before HBO2, p = 1,1 ± 10-3 at 2.5 ATA evaluation, p = 2,1 ± 10-4 for the evaluation after HBO2. The p values for r values were calculated using one tail T test (Figure 1, page 154). When comparing NPIF values obtained at each of the 10 HBO2 sessions of treatment of the same individual, we found no significant differences in homologous NPIF measurements.

We have also decided to investigate whether the values of NPIF of the patients that were submitted to HBO2 were different from those of a population of healthy volunteers (Table 2, page 153). For that we have obtained two NPIF measurements (separated by 100 minutes) in a population of healthy volunteers who were not treated by HBO2.

In this group of individuals, the mean NPIF in t = 0 min was 141,6 ± 48,3 l/min and in t = 100 min was 144,4 ± 45,7 l/min. The difference between the measurements in t = 0 min and t = 100 min was not statistically significant (p = 0,21 — using a two tailed T test for paired samples). Linear modeling was used and showed no significant variation (p = 0.48 for r value – using the two-tailed T test) (Figure 2, page 154). These data also showed that there was no significant difference between NPIF data obtained in our patients (when not under HBO2 therapy) and in healthy volunteers.

DISCUSSION

This investigation reports that when patients are under HBO2 therapy, there is a significant increase in their values of nasal inspiratory peak flow (NPIF). We also document that this enhancement is not amplified by the repetition of the HBO2 treatment, and it is transient since it sharply decreased shortly after the patients stopped the HBO2 treatment. The increase in atmospheric pressure and the enhancement in blood oxygen that characterize the HBO2 treatment are likely to contribute to the herein-reported increase in nasal peak flow values during HBO2 therapy.

Since hyperoxic vasoconstriction in the microcirculation is a local vascular response to increase in PO2 in the tissues, a certain threshold
must be achieved to trigger this mechanism, i.e., under that PO2 threshold, the vasoconstrictive effect does not occur (8). Considering a healthy nose submitted to HBO2, the underlying mechanism to an increase in the peak nasal flow may be the hyperoxic vasoconstriction of nose metarterioles and capillaries.

Our data stand on values obtained by NPIF. This is a reliable method to evaluate nasal airway patency (9). NPIF has been used, for instance, in testing the effects of drugs and in nasal allergen challenges (10).

NPIF is viewed as being as accurate as acoustic rhinometry and active anterior rhinomanometry (11,12). Clearly, the herein-reported increase in NPIF values during exposure to HBO2 have to be first associated with oxygen saturation of blood and the hyperbaric environment that are induced by staying inside the HBO2 chamber. Conceivably, the treatment will also enhance nasal mucociliary transport since this is an effect that has been previously documented in individuals under HBO2 therapy (1). Patients with nasal obstruction, particularly those with rhinitis, may have a decrease in turbinate volume, thus allowing nasal instead of oral breathing. Because nasal breathing results in the decrease in patient complaints of throat dryness and of discomfort in the middle ear or sinus, patients with nasal obstruction should be instructed to breath through the nose during the HBO2 sessions (4, 13).

Taking into account the Undersea and Hyperbaric Medical Society-approved recommendations for HBO2, it can be concluded that most individuals who are treated by HBO2 are either older or chronically ill patients who may suffer from alterations of the air pathways. For example, HBO2 is recommended for the treatment of diabetic polyneuropathy, and this disorder is associated with respiratory muscle impairment (14).

On one hand, the demonstration that HBO2 increases nasal patency indicates the therapeutic goal of HBO2 of achieving blood saturation with oxygen is facilitated by the herein-described phenomenon that will also make breathing easier for patients when they are under the HBO2 chamber conditions.

On the other hand, when patients have acute or chronic rhinosinusitis, the goal of medical or surgical treatments is to increase ventilation and drainage. The herein investigation suggests that with HBO2 that goal can also be achieved.

CONCLUSION

HBO2 improves nasal patency during the period of time that the patients are submitted to this type of therapy. Whether this advantage of HBO2 treatment will be of significance in the treatment of nasal or sinus disease is a question that deserves further investigations.

ACKNOWLEDGMENTS

The authors are grateful to Prof. Artur Águas, Instituto de Ciências Biomédicas de Abel Salazar, Universidade do Porto, Portugal, for his support, helpful comments and critical review of the manuscript and to Dr. José Brito, Faculdade de Ciências da Universidade de Lisboa for his valuable contribution on the statistics.

REFERENCES


TABLE 1. Biometrics and spirometric data of patients submitted to HBO2.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (years)</th>
<th>BMI</th>
<th>NPIF (mean) Sea level (before treatment)</th>
<th>NPIF (mean) 2.5 ATA</th>
<th>NPIF (mean) Sea level (after treatment)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>31</td>
<td>25,4</td>
<td>213,2</td>
<td>268,6</td>
<td>228,4</td>
</tr>
<tr>
<td>2</td>
<td>56</td>
<td>32,5</td>
<td>196</td>
<td>231</td>
<td>203</td>
</tr>
<tr>
<td>3</td>
<td>61</td>
<td>33,3</td>
<td>145</td>
<td>163</td>
<td>161</td>
</tr>
<tr>
<td>4</td>
<td>49</td>
<td>31,2</td>
<td>129</td>
<td>154</td>
<td>139</td>
</tr>
<tr>
<td>5</td>
<td>64</td>
<td>25,9</td>
<td>154,7</td>
<td>197</td>
<td>160</td>
</tr>
<tr>
<td>6</td>
<td>64</td>
<td>29,4</td>
<td>185,5</td>
<td>242</td>
<td>195,5</td>
</tr>
<tr>
<td>7</td>
<td>76</td>
<td>27,9</td>
<td>80,5</td>
<td>114</td>
<td>83</td>
</tr>
<tr>
<td>8</td>
<td>72</td>
<td>25,1</td>
<td>99,5</td>
<td>97</td>
<td>107</td>
</tr>
<tr>
<td>9</td>
<td>53</td>
<td>25,7</td>
<td>138</td>
<td>137</td>
<td>131</td>
</tr>
<tr>
<td>10</td>
<td>43</td>
<td>22,8</td>
<td>93,6</td>
<td>110,9</td>
<td>110,9</td>
</tr>
<tr>
<td>11</td>
<td>21</td>
<td>23,4</td>
<td>132,9</td>
<td>144,3</td>
<td>132,9</td>
</tr>
<tr>
<td>12</td>
<td>33</td>
<td>24,5</td>
<td>206,3</td>
<td>253,6</td>
<td>232,5</td>
</tr>
<tr>
<td>13</td>
<td>28</td>
<td>18,9</td>
<td>167,5</td>
<td>181,25</td>
<td>175</td>
</tr>
</tbody>
</table>
TABLE 2. Biometrics and spirometric data of the group of healthy volunteers (Vol.).

<table>
<thead>
<tr>
<th>Vol.</th>
<th>Age (years)</th>
<th>BMI</th>
<th>NPIF T = 0 min (mean)</th>
<th>NPIF T = 100 min (mean)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>53</td>
<td>29,4</td>
<td>187</td>
<td>194</td>
</tr>
<tr>
<td>2</td>
<td>36</td>
<td>25,8</td>
<td>91</td>
<td>85</td>
</tr>
<tr>
<td>3</td>
<td>46</td>
<td>33,9</td>
<td>196</td>
<td>192</td>
</tr>
<tr>
<td>4</td>
<td>52</td>
<td>27,4</td>
<td>118</td>
<td>117</td>
</tr>
<tr>
<td>5</td>
<td>38</td>
<td>26,2</td>
<td>163</td>
<td>166</td>
</tr>
<tr>
<td>6</td>
<td>25</td>
<td>22,9</td>
<td>156</td>
<td>165</td>
</tr>
<tr>
<td>7</td>
<td>32</td>
<td>23,2</td>
<td>136</td>
<td>134</td>
</tr>
<tr>
<td>8</td>
<td>26</td>
<td>23,6</td>
<td>102</td>
<td>115</td>
</tr>
<tr>
<td>9</td>
<td>57</td>
<td>38,9</td>
<td>101</td>
<td>115</td>
</tr>
<tr>
<td>10</td>
<td>57</td>
<td>24,7</td>
<td>110</td>
<td>113</td>
</tr>
<tr>
<td>11</td>
<td>53</td>
<td>24,0</td>
<td>96</td>
<td>101</td>
</tr>
<tr>
<td>12</td>
<td>47</td>
<td>24,6</td>
<td>244</td>
<td>236</td>
</tr>
</tbody>
</table>
Tables and Figures

FIGURE 1. Linear modeling of NPIF data of the HBO$_2$-treated patients.

FIGURE 2. Linear modeling of NPIF values of the group of healthy volunteers not submitted to HBO$_2$. 