



Cardiorespiratory alterations in patients undergoing hyperbaric oxygen therapy

Alterações cardiorrespiratórias de pacientes submetidos à oxigenoterapia hiperbárica

Alteraciones cardiorrespiratorias de pacientes sometidos a la oxigenoterapia hiperbárica

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-  Bruno Martinelli¹
-  Judi Meloni Noronha²
-  Maria Fernanda Misquiatti Sette²
-  Ieda Papille dos Santos³
-  Sílvia Regina Barrile¹
-  José Cláudio Simão⁴

¹ Universidade do Sagrado Coração de Bauru, Departamento de Graduação e Pós-graduação em Fisioterapia, Bauru, SP, Brazil.

² Universidade do Sagrado Coração de Bauru, Departamento de Graduação em Fisioterapia, Bauru, SP, Brazil.

³ Universidade Estadual Paulista, Faculdade de Medicina de Botucatu, Botucatu, SP, Brazil.

⁴ Faculdade Integrada de Bauru, Departamento de Graduação em Enfermagem, Bauru, SP, Brazil.

ABSTRACT

Objective: To evaluate cardiorespiratory alterations due to a single session of hyperbaric oxygen therapy. **Method:** Randomized study with patients: a control group and hyperbaric oxygen therapy. Evaluations occurred in the beginning, during, and after exposure to pure oxygen above atmosphere for 2 hours. Systemic blood pressure, peripheral oxygen saturation, pulse rate, lung volume and lung capacity, and maximal inspiratory and expiratory pressures were evaluated. Peripheral oxygen saturation, pulse rate, and systemic blood pressure were evaluated during the pressurizing in the first hour. Data were evaluated by means of ANOVA, Mann-Whitney, and independent t-test ($p < 0.05$). **Results:** A total of 14 adult patients were evaluated. In the group under therapy (seven subjects), aged: 49.57 ± 14.59 years, there was a decrease in the pulse rate of 16 beats per minute after 35 minutes of therapy (intragroup analysis), and the peripheral oxygen saturation was higher within the same period compared to the control group. **Conclusion:** The hyperbaric oxygen therapy promotes cardiorespiratory alterations with the increase of the peripheral oxygen saturation and decrease of the pulse rate, without altering blood pressure levels and the strength, volumes, and respiratory capacities.

DESCRIPTORS

Oxygen; Hyperbaric Oxygenation; Oxygen Inhalation Therapy; Arterial Pressure; Nursing Care.

Corresponding author:

Bruno Martinelli
Universidade do Sagrado Coração
Rua Irmã Arminda, 10-50, Jardim Brasil
CEP 17011-160 – Bauru, SP, Brazil
bnomartinelli@yahoo.com.br

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INTRODUCTION

Hyperbaric oxygen therapy (HBOT) is a therapeutic modality that consists of an offer of pure oxygen (O₂) by means of a single fraction of 100% O₂, in a pressurized environment, a hermetically-sealed chamber with hard walls (hyperbaric chamber), which has a pressure greater than that of the atmosphere, usually between two and three atmospheres. These chambers can accommodate one patient (*monoplace*) or many patients (*multiplace*)⁽¹⁾. “The action of this therapy is complex since it is the result of a series of physiological and pharmacological mechanisms; its “properties contribute to the treatment of several different conditions.

Nowadays, there are three types of oxygen therapy: Normobaric oxygen – surface or sea level O₂, in which there is administration of O₂ (24% to 100%), at atmospheric pressure (an atmosphere absolute – ATA); Hyperbaric Oxygen – Inhalation of 100% O₂ at elevated pressures (> than one ATA); and Hypobaric or altitude O₂ – at altitude, due to humans’ physiological limitation there is a demand for a concentration of O₂ higher than that inspired at sea level to avoid hypoxia”⁽²⁾. “This therapy is indicated for: decompression sickness or gas embolism, traumatic air embolism, gas gangrene, Fournier’s gangrene, necrotizing soft tissue infections (fasciitis, myositis, and cellulitis), acute vasculitis triggered by allergic reaction, reaction to medication or biological toxins⁽³⁾, radiation injury, acute anemia, acute traumatic ischemia among others⁽¹⁻³⁾.

Three studies have verified the hyperbaric oxygen therapy is an effective tool in the therapeutic arsenal⁽⁴⁻⁶⁾. In addition, “the benefits of the use of HBOT are described in clinical and surgical diseases with promising results, reducing hospitalization time and hospital costs”⁽³⁻⁷⁾. Absolute contraindications for HBOT use are: “only untreated pneumothorax and some chemotherapeutic agents, especially bleomycin, due to risk of pulmonary fibrosis. However, relative contraindications are: uncontrolled epilepsy, heart failure, and some airway problems, such as acute upper respiratory infection, emphysema, and previous spontaneous pneumothorax”⁽²⁾.

Side effects that can be caused due to the application of HBOT are middle ear barotrauma and gas embolism, which is the most severe complication that occurs during decompression⁽³⁾, as it can lead to respiratory system toxicity (dry cough, retrosternal pain, hemoptysis, facial discomfort, and pulmonary edema), neurological toxicity (paresthesia and seizure), hearing discomforts, and transient visual changes⁽¹⁻²⁾. Interestingly, the biochemical and cellular effects of HBOT are not completely understood; the excessive exposure of O₂ in the organism was believed to cause the worsening of the lesions, but the beneficial effects of this therapy were mainly observed during reperfusion⁽⁸⁾. Such controversies are mainly due to the lack of studies that explore biochemical, physiological, and cellular aspects⁽⁸⁻⁹⁾.

There are studies on cardiorespiratory alterations while diving, but studies on clinical disorders still need to progress, and that’s why this study is proposed. Some studies that have evaluated cardiovascular and respiratory manifestations will be presented. A study involving patients with lower limb ischemia and submitted to HBOT (2.4 ATA, 100% O₂, 90 minutes, 2 to 3 days, 1 to 3 months) demonstrated, on one hand, an increase in the airway resistance and closing volume and, on the other hand, a decrease in lung elastance, respiratory volume, respiratory rate, and vital capacity; heart rate slightly decreased⁽¹⁰⁾. Another study that proposed a 21-session treatment (24 KPa partial pressure of oxygen, 90 minutes, daily) caused a progressive reduction of lung flows and capacities during treatment. There was partial normalization four weeks after treatment. Although a decrease in conductance of small airways was observed, such effect is not considered to be clinically significant for patients treated with hyperbaric oxygen in repeated treatment sessions⁽¹¹⁾.

Deleterious effects of excessive oxygen exposure to the respiratory system were also evaluated in a study with 18 patients during 6 weeks of HBOT (daily, 90 minutes, 2.4 ATA). It was possible to verify that there was no alteration in lung volumes and capacities as well as in the capacity of diffusion of carbon monoxide⁽¹²⁾. The HBOT (2.5 ATA, 90 minutes) in 10 moderately active men did not increase the partial venous oxygen pressure, transcutaneous oxygen tension and maximal oxygen consumption. The measurements of transcutaneous and blood oxygen after the hyperbaric did not have ergogenic benefits⁽¹³⁾. In the experimental model, hyperbaric hyperoxemia has been shown to acutely induce deleterious effects on respiratory mechanics, such as the elastance and the viscoelastic components of inspiratory resistance⁽¹⁴⁾.

As shown, it is possible to conjecture that a single session of HBOT causes an increase in peripheral oxygen saturation and a reduction of cardiovascular and respiratory variables. Thus, to study the complexity and repercussions of HBOT is relevant to science, not only for its properties and for benefits, but for its indication, which becomes more frequent. Therefore, the objective of this study was to evaluate the cardiorespiratory modifications of patients submitted to a single session of HBOT.

METHOD

TYPE OF STUDY

Randomized clinical trial

POPULATION

Patients with an indication for the use of the hyperbaric chamber, Oxibarimed - Hyperbaric Medicine.

Inclusion criteria: adults, female and male, with respiratory and cardiac stability, medical indication for the application of hyperbaric oxygen therapy. Exclusion

criteria: individuals who presented difficulties in the understanding and execution of the evaluative maneuvers, physical and emotional malaise during pressurizing, thoracic drainage, hyperthermia, a history of untreated seizures, which were in the postoperative of otorhinolaryngeal and thoracic surgery, patients with spherocytosis, claustrophobia, and the pregnant women.

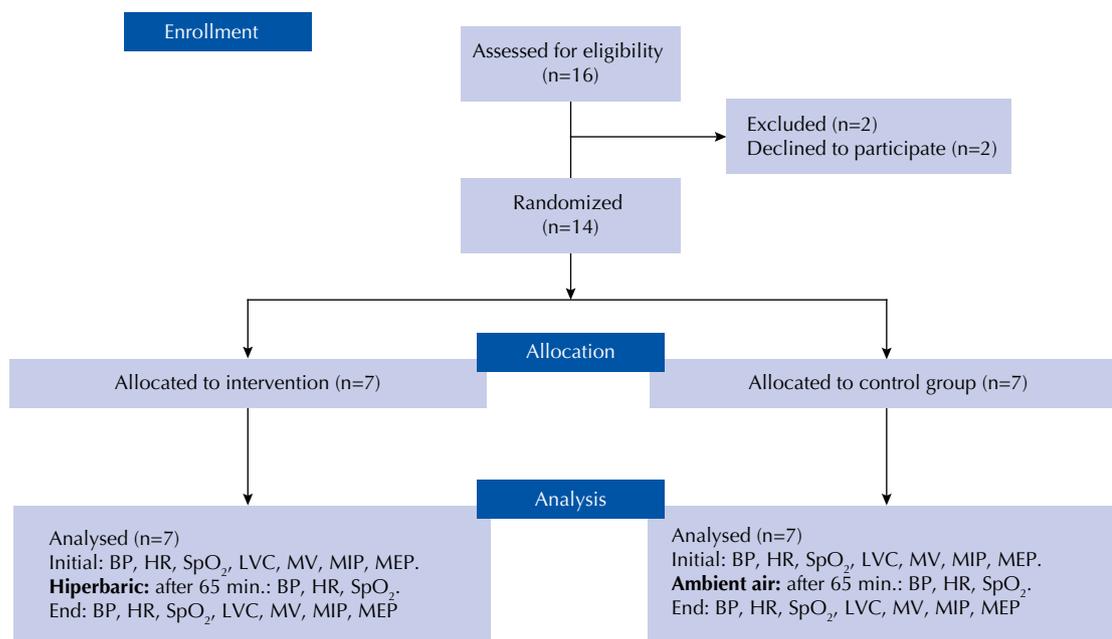
DEFINITION OF THE SAMPLE

The probabilistic sampling was composed of 16 patients with an indication for HBOT. The sample calculation was determined by a pilot study.

Patients were divided in two groups: control (without pressurization, i.e., without HBOT, staying in a specific room with ambient air) and group under pressurization (HBOT).

DATA COLLECTION

Both groups were evaluated at three moments: without pressurization (basal), after 35 minutes, and after 120 minutes (final). The evaluation after 35 minutes of therapy was defined because of the moment corresponding to the interval stipulated by the monitoring team. At that moment, it was possible to observe systemic blood pressure, peripheral oxygen saturation, and pulse rate. After a five-minute interval, there was continuous use of O₂ again (Figure 1).



Caption: BP= blood pressure (mmHg), HR= heart rate (bpm), SpO₂= peripheral oxygen saturation (%), LVC= Low vital capacity (l), MV= minute volume (l/min), MIP= maximum inspiratory pressure (cmH₂O), MEP= maximum expiratory pressure (cmH₂O).

Figure 1 – Flow chart with the stages of the study and the analysis of patients with an indication of HBOT.

Body mass index (BMI) was calculated based on the formula weight/height (Kg/m²) to obtain the body size of volunteers according to the *World Health Organization*⁽¹⁵⁾.

Physical activity was classified as regular, irregular, and sedentary according to the definitions: regular – physical exercises in the free time three or more times per week; irregular – physical exercises up to two times per week; sedentary – no physical exercise.

In addition, cardiorespiratory parameters were measured. Patients were positioned seated in a comfortable position, resting for 5 minutes. To check systemic blood pressure (BP), systolic (SBP) and diastolic (DBP) mmHg, we used an automatic digital arm blood pressure monitor (model 2005 – Bioland Technology®, China – INMETRO ML 01602010) and followed the guidelines of the Brazilian Society of Cardiology (*Sociedade Brasileira de Cardiologia*),

Brazilian Society of Hypertension (*Sociedade Brasileira de Hipertensão*), and Brazilian Society of Nephrology (*Sociedade Brasileira de Nefrologia*)⁽¹⁶⁾.

Pulse rate (PR – bpm) and peripheral oxygen saturation (SpO₂ – %) were checked by the adult finger pulse oximeter PM100C (New Tech®, EUA), positioned on the fifth finger of the hand.

Slow Vital Capacity (SVC) and volume per minute (VM – L/min) were measured by the Mark 8 Wright analog respirometer (Ferraris®, United Kingdom), with a one-minute interval between trials. CV maneuver was performed by deep inspiration to total lung capacity and, right in the sequence, by a slow and maximum expiration until the residual volume⁽¹⁷⁻¹⁸⁾.

We used an analog pressure gauge (Comercial Médica®, Brazil) with a scale between 0 and 120 cmH₂O to measure

the maximum respiratory pressures and a nose clip to avoid air leakage. Maximum inspiratory (MIP) and expiratory (MEP) pressures were measured with maneuvers performed between the Residual Volume (RV) and the Total Vital Capacity (TVC)⁽¹⁹⁻²⁰⁾.

Patients who underwent hyperbaric oxygen therapy were positioned in a multiplace hyperbaric chamber A240 (SeawayDiver®, Brazil), which is a pressurized ambient under pressure greater than an absolute atmosphere (approximately 2.5 ATA) with intermittent ventilation of pure oxygen (100%). This session occurred in the morning and lasted 2 hours. After the patients had been positioned in the

chamber, pressurization took about 15 minutes (six meters). They should put on the oxygen masks and stay with it for 50 minutes (15 meters – 2.5 ATA). In the sequence, there was a scheduled 5-minute interval in which the mask was taken so the patients could drink water if necessary or desired. After this interval, the mask was placed again and patients were given 100% oxygen, for an additional 45 minutes, until the moment of the depressurizing. Patients used the mask for other 5 minutes until depressurizing by three meters and then removed the mask. The chamber was fully depressurized for a period of approximately 10 minutes; patients were then released. This process is illustrated in Figure 2.

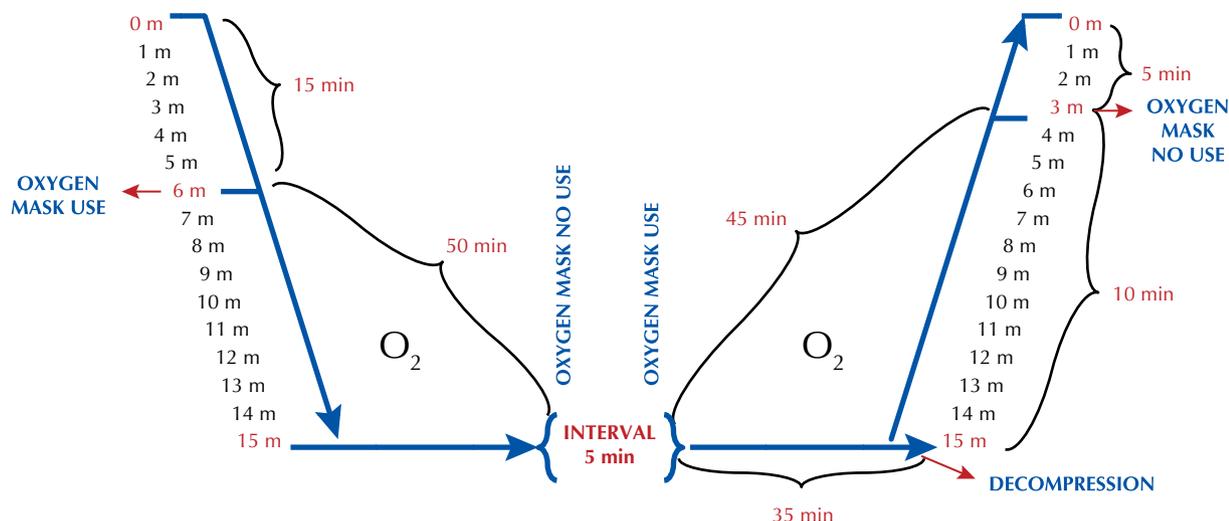


Figure 2 – Diagram illustrating the pressure (atmospheres absolute) and time of the hyperbaric chamber.

DATA ANALYSIS AND TREATMENT

Descriptive data were presented with the absolute and relative distribution. The Shapiro-Wilk test was used to perform the analysis of data distribution. Regarding the comparative analysis, intragroup – baseline, after 35 minutes and final, the ANOVA with repeated measures was used; consequently, for the comparative analysis, intergroup, independent t-test (normal distribution) and Mann Whitney test (non-normal distribution) were used. The central tendency measures are average, standard deviation, and median (interquartile range), respectively ($p < 0.05$). The software used was the Sigma Test, 12.0.

ETHICAL ASPECTS

This study was approved by the Research Ethics Committee of the Universidade do Sagrado Coração, on the 22nd of April, 2015, under n. 1.031.237. It complied with the precepts of the Resolution n. 466/12 of the National Health Council.

RESULTS

A total of 14 patients were evaluated, six (42.85%) women and eight (57.15%) men, aged 55.2 ± 14.29 years,

with a weight of 80.5 ± 21.6 kg and a height of 1.71 ± 0.12 m. Regarding ethnicity, seven (50%) were white and seven (50%) were brown. The main diagnostic hypotheses were a venous ulcer, osteomyelitis, postoperative of general surgery, and Fournier's syndrome. Regarding social habits, three (21.42%) were smokers, one (7.14%) former smoker, two (14.28%) regularly practiced physical activities, two (14.28%) irregularly practiced physical activities, and the others were sedentary.

Specifically, the control group ($n=7$) had four (57.15%) men, with a weight of 82.14 ± 20 kg and a height of 1.66 ± 0.11 m. Most individuals in this group were white, only one (14.28%) was brown; one (14.28%) was a smoker, and one (14.28%) was a former smoker, only two (28.57%) regularly practiced physical activities, and one (14.28%), irregularly. In the group under therapy ($n=7$), there were four men (57.15%), with a weight of 78.8 ± 24.54 kg and a height of 1.76 ± 0.11 m. Most individuals in this group were brown, only one (14.28%) was white. In this group, two (28.57%) were smokers and the others weren't; only one (14.28%) regularly practiced physical activities. Table 1 shows the basal data collected from both groups.

Table 1 – Description of the baseline anthropometric and cardiorespiratory characteristics from both groups – Bauru, SP, Brazil, 2016.

Variable	Group	
	Control (n=7)	HBOT (n=7)
Age (years)	60.85±12.48	49.57±14.59
BMI (Kg/m ²)	29.55±6.45	24.80±4.39
SBP (mmHg)	138.71±40.96	139.14±25.90
DBP (mmHg)	76.85±24.39	85.42±18.40
SpO ₂ (%)	94.14±4.59	94.71±2.36
HR (bpm)	78.28±16.54	83.42±8.20
RR (bpm)	21.14±4.59	16.00±5.56
MV (l/min)	16.81±8.81	20.81±7.06
LVC (l)	4.34±0.88	5.20±1.13
MIP (cmH ₂ O)	61.71±31.14	81.71±41.23
MEP (cmH ₂ O)	53.71±31.98	82.28±34.93

Caption: HBOT= hyperbaric oxygen therapy; BMI= body mass index; SBP= systolic blood pressure; DBP= diastolic blood pressure; SpO₂= peripheral oxygen saturation; HR= heart rate; RR= respiratory rate; MV= minute volume; LVC= low vital capacity; MIP= maximum inspiratory pressure; MEP= maximum expiratory pressure.

Statistical analyses showed there was no significant difference between the analyzed groups.

Regarding the intragroup analysis, there was no statistically significant difference in the control group for any of the variables studied; however, in the group under therapy, there was a difference in the pulse rate in the comparison between baseline and 35 minutes of therapy. Table 2 shows the variables in the three moments of the group under therapy (hyperbaric).

Table 2 – Results of the peripheral oxygen saturation and cardiac variables in the initial moment, after 35 minutes, and in the final moment of the group under therapy – Bauru, SP, Brazil, 2016.

Variables	Time		
	Initial	35 min	Final
SBP (mmHg)	139.14±25.9	122.85±29.27	135.714±29.33
DBP (mmHg)	85.42±18.40	88.57±26.09	77.429±18.34
HR (bpm)	83.42±8.20	66.57±14.92*	72.143±9.08
SpO ₂ (%)	94.71±2.36	98.57±0.53	95.571±5.09

Caption: * comparison between initial and 35 min. time (p<0,05); SBP=systolic blood pressure; DBP=diastolic blood pressure; HR=heart rate; SpO₂=peripheral oxygen saturation.

Table 3 shows the respiratory variables evaluated in the initial and final moments on the group under therapy.

Table 3 – Results of the respiratory variables in the initial and final time of the group under therapy – Bauru, SP, Brazil, 2016.

Variable	Time	
	Initial	Final
RR (bpm)	16.00±5.56	17.71±7.25
MV (l/min)	20.81±7.06	25.05±15.38
VC (l)	5.20±1.13	5.15±1.38
MIP (cmH ₂ O)	81.71±41.23	90.28±34.00
MEP (cmH ₂ O)	82.28±34.93	84.00±26.12

Caption: RR=respiratory rate; MV=minute volume; VC=low vital capacity; MIP=maximum inspiratory pressure; MEP=maximum expiratory pressure.

It is possible to notice that there was no alteration of the respiratory variables in the comparison between the initial and final moments in the group under therapy.

In the intergroup analysis, there was differentiation in the SpO₂ variable (%) after 35 minutes of therapy; in the control group, it was 95.00 (90.00–98.00) and in the therapy group, 99.00 (98.00–99.00).

Regarding clinical manifestations, only one patient (7.14%) reported obnubilation during the tests; however, after a few minutes of rest, it was possible to continue the data collection.

DISCUSSION

This study verified there is an alteration of the peripheral O₂ saturation and pulse rate under the influence of hyperbaric oxygen therapy in patients with distinct clinical impairments. The most important findings are discussed below. On one hand, since the body is submitted to hyperbaric pressure, the tissues receive a large supply of O₂ that is bound to the hemoglobin molecule and, mainly, there is dissolution in the blood plasma. On the other hand, if only total hemoglobin saturation is considered, i.e., 100%, this condition could be verified without any pressure modification⁽²¹⁾. According to this statement, an increase of SpO₂ was expected since the procedure was performed in a pressurized ambient under a pressure greater than an absolute atmosphere with pure O₂ ventilation (100%).

In the HBOT, tissues of lower metabolism have a greater decrease in blood flow, a vasoconstrictor effect. “In most tissues, blood flow decreases in tissue almost proportionally to basal consumption. There is generalized vasoconstriction, with the exception of pulmonary circulation since hyperbaric hyperoxia causes significant vasodilation of the pulmonary vessels after 15 minutes of therapy. There is a noticeable increase in the tension of O₂ in all body fluids under these conditions. About 6.4 ml of O₂ are dissolved in every 100 ml of blood, in addition to the hemoglobin-bound content, which exposes the organism to an ambient pressure higher than normal. The partial pressure of the

gases in the pulmonary alveoli increases proportionally, from the partial pressures of O₂, until reaching hyperbaric hyperoxia. This increase in the content of the arterial O₂, in percentage increases, for various concentrations of hemoglobin; compared to conditions of normoxia and hyperbaric hyperoxia (three ATA), hemoglobin in the venous blood still remains practically saturated for some time after, which explains the increase in peripheral saturation observed in this study^(13,21).

Within this context, it is possible to present the benefits and harms of O₂. The oxygen "is considered a drug that can be easily administered under normobaric conditions. In addition, the medical O₂ is the most widely used gas in the medical and emergency areas; this medical gas is considered a pharmaceutical product and if there is a failure in this supply of O₂, it is necessary to resort to its therapeutic application. Damagingly, the inhalation of high doses of O₂ can increase the formation of free radicals that can lead to oxidation of tissue chemical components^(2,22), oxidative damage of deoxyribonucleic acid (DNA), and a worsening in the rate of cell death⁽⁵⁾. The benefits of hyperbaric oxygen therapy are derived from the physiological and pharmacological effects of O₂ in high doses. These were classified as systemic effects of the HBOT: a depression of the activities of the carotid and aortic receptors, an increase in arterial content of O₂, bradycardia, a decrease of cardiac output and peripheral vasoconstriction, and an increase of the systemic vascular resistance⁽²³⁾. Physiologists have identified bradycardia when the human body undergoes pressure changes⁽²⁴⁾.

In the current study, bradycardia was verified after 65 minutes of therapy in 15 meters (2.5 ATA) and after 50 minutes of exposition to 100% O₂. This finding corroborates with other pieces of evidence. Responses of the cardiovascular system to hyperbaric hyperoxia were verified: vasoconstriction, hypertension, and a decrease in the heart rate and, consequently, in the cardiac output. Initially, these responses at moderate levels of hyperbaric hyperoxia are coordinated by baroreflex mechanism mediated by vasoconstriction. Furthermore, baroreceptor activation inhibits sympathetic outflow and may partially reverse an O₂ – dependent increase in blood pressure⁽²⁵⁾. The explanations for these phenomena have been detailed. The heart rate modulation was analyzed during hyperbaric pressure in 10 divers exposed to one, two, three, and four ATA. Bradycardia was confirmed with the increase in pressure; it is interesting to mention that the fall in HR reached statistical significance after two ATA, i.e., That is, increased pressure caused an increase in bradycardia, and cardiac modulation predominated in the high-frequency, parasympathetic⁽²⁶⁾.

In another study, motivated by the information that exposure to supranormal O₂ pressures induces bradycardia and peripheral vasoconstriction, four situations were created with and without a hyperbaric treatment at different pressures (one and 2.5 ATA) and inspired fractions of O₂ (21% and 100%) to be tested on healthy volunteers. Again, HR decreased during all interventions, but with no

difference between sessions. The data suggest that hyper and normobaric hyperoxia increases the parasympathetic influx in cardiac regulation⁽²⁷⁾.

In the following year, the same authors conducted research again, but this time with professional divers, maintaining both situations: 100% hyperbaric oxygen in 2.5 ATA and 21% hyperbaric air in 2.5 ATA. HR decreased, but the response was similar in both treatments. There were no alterations in cardiac conduction or incidence of arrhythmias; however, 100% O₂ at 2.5 ATA caused a marked increase in the parasympathetic tone⁽²⁸⁾. Within an animal model, the effect from one to five bar of O₂ in conscious and anesthetized rats was studied. Exposure to O₂ stimulates the myocardium by elevating left ventricular pressure and pulse pressure. The arrhythmia condition was observed in both groups; however, bradycardia occurred only in the state of consciousness⁽²⁹⁾.

In contrast, a literature review that compared normal and hyperbaric ambient verified the heart rate ($p = 0.1468$; > 0.05) showed a significant difference between the verified types of ambient. The maximum O₂ consumption ($p = 0.00013$; < 0.05) showed significant differences between these environments, though⁽³⁰⁾. It is evident that the exposure of the biological system (healthy, ill or conditioned) to hyperbaric pressure changes the cardiac control, which can be verified by the decrease of the heart rate.

In the current study, this reduction still occurred within the limits of normality and did not cause other clinical symptoms. It was expected that there would be no changes in the variables studied in the control group since it was not influenced by HBOT and, consequently, the cardiorespiratory variables remained stable. The control group is important due to the fact that it allows the comparison between the groups and to provide equal chances of all patients being submitted to HBOT. This study was limited to the heterogeneous sample regarding the pathology and the acute condition of the HBOT. Further studies are suggested to allow a specific evaluation of each disease condition and in the long term. Up to the findings of the present research and our knowledge, there is no study with this approach providing relevant information to the health team about the repercussions of this therapy, providing subsidies for prescription and monitoring. Therefore, it is possible to accept the hypothesis that a single HBOT session causes an increase in the peripheral oxygen saturation and a decrease in the pulse rate in patients with vascular, surgical, and inflammatory disorders.

CONCLUSION

Considering the facts mentioned in the discussion, we conclude that HBOT promotes significant cardiorespiratory changes with increased peripheral oxygen saturation and reduced pulse rate.

RESUMO

Objetivo: Avaliar modificações cardiorrespiratórias em decorrência de sessão única de oxigenoterapia hiperbárica. **Método:** Estudo aleatorizado com pacientes: grupos-controle e oxigenoterapia hiperbárica. As avaliações ocorreram no início, durante e após a exposição ao oxigênio puro acima de uma atmosfera, durante 2 horas. A pressão arterial sistêmica, saturação periférica de oxigênio, frequência de pulso, volume e capacidade pulmonar, pressões inspiratória e expiratória máximas foram avaliadas. A saturação periférica de oxigênio, frequência de pulso e pressão arterial sistêmica foram avaliadas durante a pressurização na primeira hora. Os dados foram avaliados pelo teste de ANOVA, Mann-Whitney e teste t independente ($p < 0,05$). **Resultados:** Foram avaliados 14 pacientes adultos. No grupo sob terapia (sete sujeitos), idade: $49,57 \pm 14,59$ anos houve redução da frequência de pulso de 16 batimentos por minuto após 35 minutos de terapia (análise intragrupo), e a saturação periférica de oxigênio foi maior neste mesmo período se comparado ao grupo-controle. **Conclusão:** A oxigenoterapia hiperbárica promove alterações cardiorrespiratórias com o aumento da saturação periférica de oxigênio e redução da frequência de pulso, sem alterar os níveis pressóricos arteriais e a força, volumes e capacidades respiratórios.

DESCRITORES

Oxigênio; Oxigenação Hiperbárica; Oxigenoterapia; Pressão Arterial; Cuidados de Enfermagem.

RESUMEN

Objetivo: Evaluar modificaciones cardiorrespiratorias consecuentes de sesión única de oxigenoterapia hiperbárica. **Método:** Estudio aleatorizado con pacientes: grupos de control y oxigenoterapia hiperbárica. Las evaluaciones ocurrieron en el inicio, durante y después de la exposición al oxígeno puro por encima de una atmósfera, durante dos horas. La presión arterial sistémica, saturación periférica de oxígeno, frecuencia de pulso, volumen y capacidad pulmonar, presiones inspiratoria y expiratoria máximas fueron evaluadas. La saturación periférica de oxígeno, frecuencia de pulso y presión arterial sistémica fueron evaluadas durante la presurización en la primera hora. Los datos fueron evaluados por el test de ANOVA, Mann Whitney y prueba t independiente ($p < 0,05$). **Resultados:** Fueron evaluados 14 pacientes adultos. En el grupo bajo terapia (siete sujetos), edad: $49,57 \pm 14,59$ años, hubo reducción de la frecuencia de pulso de 16 latidos por minuto tras 35 minutos de terapia (análisis intragrupo), y la saturación periférica de oxígeno fue mayor en ese mismo período si comparado con el grupo de control. **Conclusión:** La oxigenoterapia hiperbárica proporciona alteraciones cardiorrespiratorias con el aumento de la saturación periférica de oxígeno y la reducción de la frecuencia de pulso, sin alterar los niveles de presión arteriales y la fuerza, volúmenes y capacidades respiratorios.

DESCRIPTORES

Oxigênio; Oxigenación Hiperbárica; Terapia por Inhalación de Oxígeno; Presión Arterial; Atención de Enfermería.

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