Hemodynamic Modifications During Hyperbaric Oxygen Therapy

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Pelaia P, Rocco M, Conti G, De Blasi RA, Bufi M, Antonelli M, Bortone C. Hemodynamic modifications during hyperbaric oxygen therapy. J Hyperbaric Med 1992; 7(4):229–237.— Hyperbaric oxygenation (HBO) can determine some hemodynamic modifications. We studied in 10 volunteers, by means of the thoracic bio-impedance, the relationship between these hemodynamic modifications and standard hyperbaric therapy based on intermittent oxygen cycles. To validate this noninvasive method in the hyperbaric chamber, we compared the agreement of cardiac output measured with this device with the cardiac output measured by thermodilution at 2.0 atm abs. We found a significant correlation coefficient and a good agreement measured by bias. During 100% oxygen 25-min cycles we recorded the following parameters in 10 subjects: heart rate (HR), stroke volume (SV), cardiac output (CO), thoracic fluid index (TFI), and blood pressure (MAP). We verified that HBO induced a decrease in CO values ($P < 0.05$), in SV values ($P < 0.05$), and in HR ($P < 0.001$), whereas MAP increased ($P < 0.01$). These effects became evident in the second cycle, reached maximum levels during the third cycle, and did not return to basal values during air intervals. The results led us to look at the exposure time at this PpO_2 instead of the hyperoxia by itself in inducing CO modifications.

hyperbaric oxygenation, hemodynamic, thermodilution, thoracic bio-impedance

Introduction

Thoracic bio-impedance (1, 2) has gained popularity as a noninvasive method for the estimation of stroke volume and cardiac output, providing some clinically useful information on patients where the risks of pulmonary artery catheterization outweigh the potential benefits (3, 4). It is well known that hyperbaric oxygenation (HBO) determines some hemodynamic modifications, described in the last years in many animal studies (5–8).

Pisarello and co-workers (9) studied human circulatory responses to prolonged hyperbaric hyperoxia. They measured in healthy volunteers the effects of breathing 100% oxygen continuously at 3.0, 2.5, 2.0 atm abs for many hours on a cardiovascular system, finding a decrease of the cardiac output (CO) in the early phase that recovered in the late period, related to heart rate changes.

The aim of the present study was to determine by means of a thoracic bio-impedance device the relation between the hemodynamic modifications

previously reported and a standard hyperbaric therapy, based on intermittent oxygen cycles in a group of 10 volunteers. To validate the thoracic bio-impedance technique in the hyperbaric chamber we simultaneously compared the agreement of CO measured with this device (CObi) with the CO measured by thermodilution (COtd).

Validation of the Technique

Clinical studies (10–12) have shown that CO values measured by thoracic bio-impedance (CObi) has a good correlation with the thermodilution technique (COtd) ($r = 0.83/0.92$). To verify whether the higher pressure present in the hyperbaric chamber could interfere with the bio-impedance device, we made simultaneous CO measures with this device and a pulmonary artery catheter. A politrauma critically ill patient who required a pulmonary artery catheter for clinical management and HBO therapy for cerebral edema entered this validation protocol. The patient, a male 34 yr old, had no pacemaker, dysrhythmias, or tachycardia (10–13), and he was in a stable hemodynamic condition. We made nine CO measurements at 2.0 atm abs with NCCOM3 BOMED Medical Instruments (USA) and by injecting 10 ml of D5W at room temperature with Edwards pulmonary artery catheter (Santa Ana, CA) and Hewlett Packard 78551D CO computers (Palo Alto, CA). To avoid possible technical mistakes, the same operator made the fluid injections simultaneously to the CObi paper recording.

We compared the agreement of CObi with COtd statistically with correlation coefficient and bias (the mean difference between two methods). The results are shown in Table 1. The COtd showed mean values of 7.57 + 0.82 liter/min and the CObi showed mean values of 6.6 + 0.5 liter/min with a significant correlation: $r^2 = 0.928$ $P < 0.01$ (Fig. 1) and a good agreement measured by bias (Fig. 2). The bias represents the systematic error between two measurement techniques, and Bland and Altman (14) have proposed that bias, the

Table 1: Cardiac Output Measures, td/bi

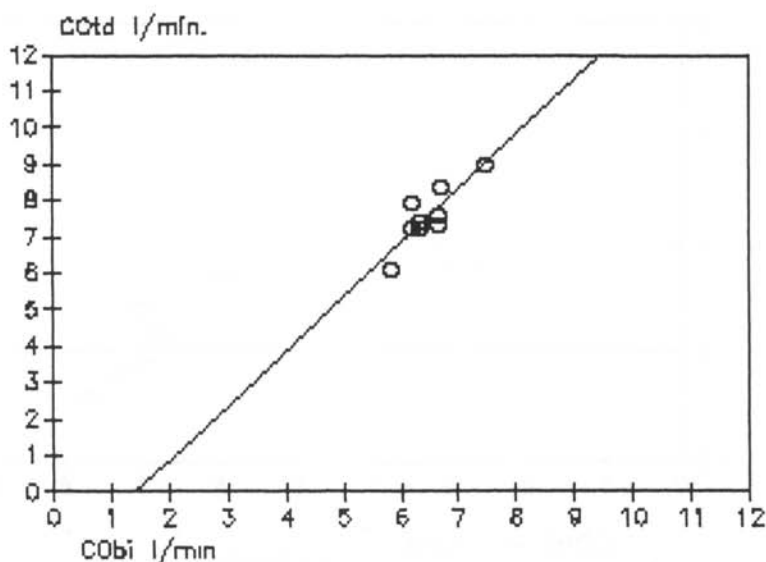| Samples	COtd	CObi
1	7.3	6.7
2	6.1	5.8
3	7.2	6.3
4	7.2	6.4
5	7.4	6.4
6	7.9	6.7
7	7.6	6.5
8	9.0	7.6
9	8.4	7.0

FIG. 1—Linear regression COtd/CObi.

mean difference between two methods, rather than correlation and regression, more correctly compares two methods that measure the same parameter. This method may be used to analyze the repeatability of a single measurement method or to compare measurements by two observers.

This correlation between the two techniques demonstrates that the pressure present in the hyperbaric chamber does not interfere with the reliability of CO measured with the bio-impedance technique.

Patients and Method

In our protocol we studied 10 normal, informed subjects, mean age 35 ± 5.7 yr, suitable for HBO therapy. We utilized a cardiovascular monitor NCCOM3 BOMED able to measure noninvasively by the electric bio-impedance technique the following hemodynamic parameters: heart rate (HR), stroke volume (SV), CO, and thoracic fluid index (TFI).

We recorded blood pressure with a Dynamap 8101 (Ethicon) at regular intervals. We then calculated the systemic vascular resistance (SVR), cardiac index (CI), and mean arterial pressure (MAP). All these parameters were recorded with an alpha numeric printer. All the subjects were pressurized at 2.2 atm abs and breathed 100% oxygen for three 25-min cycles with 5-min air intervals. The times of sampling are shown in Table 2.

The results were analyzed by analysis of variance one-way repeated test and P values <0.05 with the Tukey test to evaluate the correlation between the different recording times.
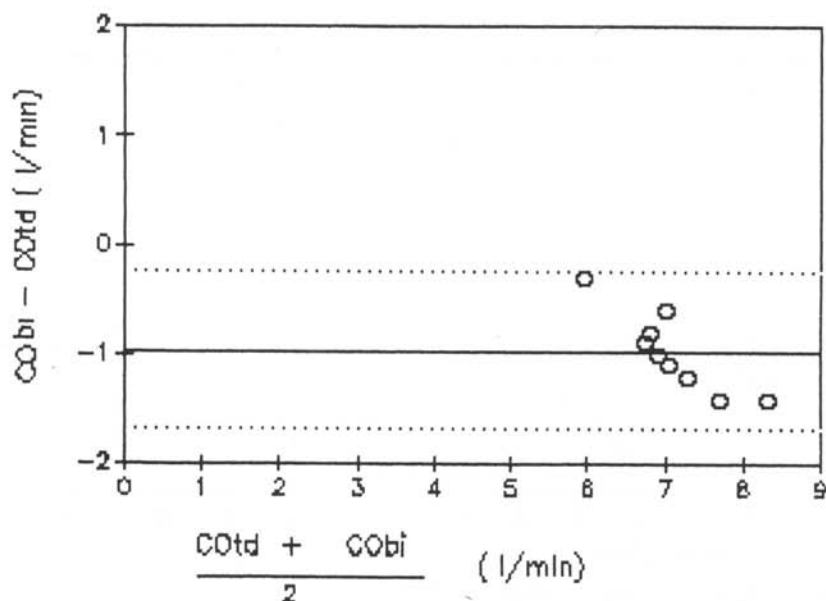
FIG. 2—Measures agreement by bias.

Table 2. Times of Sampling, min

T0	basal	
T1	hyperbaric oxygen	10
T2	hyperbaric oxygen	20
T3	air	5
T4	hyperbaric oxygen	10
T5	hyperbaric oxygen	20
T6	air	5
T7	hyperbaric oxygen	10
T8	hyperbaric oxygen	20
T9	decompression	3
T10	decompression	5
T11	recovery	25
T12	recovery	30
T13	recovery	55

Results

The results are shown in Table 3. We registered a decrease in CO values from 7.48 to 5.79 liter/min (T4) that increased to 6.60 at the end of the study ($P < 0.05$); SV decreased from 104.9 to 95.7 ml; HR decreased from 73 to

Table 3: Hemodynamic Modifications During HBO

	B	T1	T2	T3	T4	T5	T6	T7	T8	T9	T10	T11	T12	T13
CO, liter/min	7.48±1	6.2±1.6	6.1±1.4	6.1±1.2	5.8±1.6	5.9±1.6	6.2±1.5	6.3±1.5	6.1±1	6.4±1.3	6.7±2.2	0.6.7±1	6.6±1.3	6.6±1.2
SV, ml	104±29	99.7±25	97.5±22	91.7±19	97.1±24	94.8±27	93.2±20	100±25	94±29	107±26	122±69	108±27	109±29	95.7±23
HR, beats/min	73±10.8	63.3±10	64.8±11	65.1±12	60.7±11	64±12.2	65.5±9	63±10.2	65.8±9	61.5±10	63±10.6	63±9.7	62.8±12	67.6±9.5
MAP, mmHg	96.7±9	93.5±8	88.6±12	94.6±7	92±9.66	94.7±7	91.7±11	98.7±4	107±6	90.4±9	90.7±8	91.2±9	91±10.5	94.4±7.1
TFI, ohms	20.6±2	20.9±2	21.6±2	21.3±2	21.5±2	21.3±2	21.3±2	21.2±2	21.5±2	21.6±2	21.5±2	21.4±2	21.5±2	51.5±2.7

60.7 beats/min returning to 67.6 at the end of the protocol ($P < 0.001$), and the MAP changed from 96.7 to 88.6 (T2) to 107.7 mmHg (T8) returning to 94.4 mmHg (T13) ($P <0.01$) (Fig. 3).

The other parameters studied remained unchanged. As shown in Table 3, HBO effects on the cardiovascular system are not evident in the first O_2 cycle but become evident in the second, reaching a maximum level during the third cycle. These hemodynamic parameters did not return to the basal values during air intervals (5 min). Moreover we did not observe a normalization of these parameters at 55 min after the end of the treatment.

Discussion

The thoracic bio-impedance technique measures the CO noninvasively by placing two pairs of ECG electrodes on the neck and two pairs on the mid-axillary line at the level of the xiphoid process. This technique calculates the pulsatile change in resistance to injected microcurrents, 70 kHz, 2.5 mA, into the thoracic tissue. This resistance depends on the fluid characteristics of the thoracic volume, and is timed to ventricular depolarization and systole and
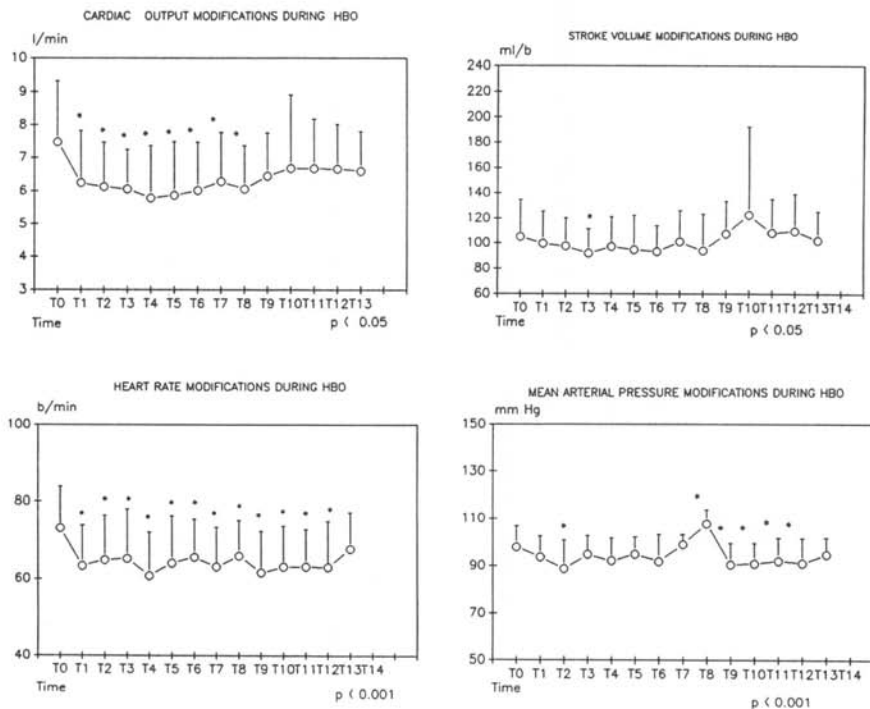

FIG. 3—Hemodynamic modifications during HBO.

can be used to calculate the SV according to the Bernstein equation (1, 2). Clinical studies have demonstrated that CO calculated by bio-impedance (CObi) is highly correlated to COtd (1–11). Moreover Wong et al. (15) demonstrated the reproducibility ($>97.9\%$) of these noninvasive measures as they obtained them by thermodilution.

We demonstrated that the pressure present in the hyperbaric chamber does not interfere with the reliability of CO measured with the bio-impedance technique. In fact, the statistical analysis shows a significant correlation between the CO measured with these two techniques in the hyperbaric chamber and a good agreement evaluated by the bias between CObi and COtd. In the present study we evaluated the reliability of the two different methods of measuring the same parameter by correlation and bias as proposed by Bland and Altman (14). For many years it has been well documented that oxygen breathing in man is limited by toxic effects that become more severe as inspired oxygen and exposure duration are increased.

Many animal studies (5–17) show that HBO decreases coronary blood flow (CBF) and CO because of a marked vasoconstriction. Many authors (5–18) have shown that CO decreases in proportion to the increase in the SVR, although the degree of vasoconstriction differs greatly. The different vascular beds respond differently to the same increased O_2 tension. The blood flow to the kidney, the adrenals, and liver are unchanged, whereas the left and right heart ventricle blood flow (LVBF/RVBF) decreases by 41 and 47%, respectively (5). Moreover, Hordnes and Tyssebotn (8) showed that the effect on CO was independent of the ambient pressure but well correlated to the Po_2. Savitt et al. (16) affirmed that the CBF and CO decrease with HBO did not seem to be associated with primary alterations in myocardial energetic function.

Although the CO decreasing mechanism has not been defined, the flow reduction determines a decreasing O_2 supply disproportionate to the pump work of the heart. From the physiology we know that the myocardium has little possibility of increasing O_2 extraction, and this discrepancy between O_2 heart demand and supply can determine the myocardial hypoxia. Pisarello et al. (9) studied the cardiocirculatory modifications to prolonged hyperbaric hyperoxia. The volunteer group studied breathing 100% oxygen continuously at 3.0, 2.5, and 2.0 atm abs for many hours. They observed an initial decrease in CO that partially recovered in the latter part of the exposure, related to HR variations. In this present study we evaluated how a therapeutic hyperbaric cycle with an intermittent and not a continuous oxygen exposure interferes with the hemodynamic parameters. In the group of healthy volunteers studied, CO values decreased significantly in the first cycle, reaching the lowest level during the second O_2 cycle. These values did not return to basal levels during air breathing intervals (5 min) and 55 min after the end of the treatment. The HR showed a decrease, in general parallel to the CO variations; the SV values, related to CO and HR remained unchanged, guaranteeing stable hemodynamics.

Until today we have applied a therapeutic hypothesis from Lambertsen (18) that provides for the application of air intervals to raise the oxygen tolerance, with special reference to pulmonary toxicity. In their paper, Pisarello et al. (9) proved the presence of a sort of "adaptation" with the recovery of HR and CO in the late exposure. In our study, 5-min air intervals have not been sufficient for recovery, on the contrary preventing the appearance of these adaptation mechanisms. These results led us to direct our attention to the exposure time at these Pp_{O_2} instead of the hyperoxia by itself in determining CO modifications.

In conclusion, we recognize the importance of monitoring the hemodynamic parameters of the patients considered at risk for cardiocirculatory problems, in whom modifications previously described could cause unstable problems.

References

1. Bernstein DP. Continuous noninvasive real time monitoring of stroke volume and cardiac output by thoracic bioimpedance. Crit Care Med 1987; 14:898–901.
2. Bernstein DP. A new stroke volume equation for thoracic electrical bioimpedance: theory and rationale. Crit Care Med 1986; 14:902–904.
3. Robin ED. (editorial) Death by pulmonary artery flow-directed catheter: time for a moratorium. Chest 1987; 92:727–731.
4. Patel C, Laboy V, Venus B, Mathru M, Weir D. Acute complication of pulmonary artery catheter insertion in critically ill patients. Crit Care Med 1986; 14:195–197.
5. Bergo GW, Risberg J, Tyssebotn I. Effect of 5 bar oxygen on cardiac output and organ blood flow in conscious rats. Undersea Biomed Res 1989; 15:457–470.
6. Risberg J, Tyssebotn I. Hyperbaric exposure to a 5 ATA He-N_2-O_2 atmosphere affects the cardiac function and organ blood flow distribution in awake trained rats. Undersea Biomed Res 1986; 13:77–90.
7. Sventek JC, Ambraski EJ. Effects of 100% oxygen on the cardiovascular responses to vasoactive compounds in the dogs. Aviat Space Environ Med 1985; 56:972.
8. Hordnes C, Tyssebotn I. Effect of high ambient pressure and oxygen tension on organ blood flow in conscious trained rats. Undersea Biomed Res 1985; 12:115–128.
9. Pisarello JB, Clark JM, Lambertsen CJ, Gelfand R. Human circulatory responses to prolonged hyperbaric hyperoxia in predictive studies V. In: Bove AA, Bachrach AJ, Greenbaum LJ Jr, eds. Underwater and hyperbaric physiology. Proceedings of the ninth international symposium on underwater and hyperbaric physiology. Bethesda MD: Undersea and Hyperbaric Medical Society, 1987.
10. Appel PL, Kram HB, MacKabel J, et al. Comparison of measurements of cardiac output by bioimpedance and thermodilution in severely ill surgical patients. Crit Care Med 1986; 14:933.
11. Saladin V, Zussa C, Risica G, et al. Comparison of cardiac output estimation by thoracic electrical bioimpedance, thermodilution and Fick methods. Crit Care Med 1988; 16:1157.
12. Wong DH, Tremper K, Stemmer E, O'Connor D, Wilbur S, Zaccari J, et al. Noninvasive cardiac output: simultaneous comparison of two different methods with thermodilution. Anesthesiology 1990; 72:784–792.
13. Spinale FG, Reines HD, Crawford FA. Comparison of bioimpedance and thermodilution cardiac output: experimental and clinical studies. Ann Thorac Surg 1988; 45:421–425.
14. Bland MJ, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. Lancet 1986; 2:307–310.

15. Wong D, Onishi R, Tremper K, Reeves C, Zaccari J, Wong A. Thoracic bioimpedance and Doppler cardiac output measurement: learning curve and interobserver reproducibility. Crit Care Med 1989; 17:1194–1198.
16. Savitt MA, Elbeery JR, Owen CH, Rankin JS, Camporesi EM. Mechanism of decreased coronary and systemic blood flow during hyperbaric oxygenation. Undersea Biomed Res. 1989; 16(suppl):50–51.
17. Pelled B, Seki Y, Ramsey F, Lambertsen CJ. Effects of hyperoxia on the coronary circulation and myocardial function. In: Trapp WG, Banister EW, Davison AJ, Trapp PA, eds. Proceedings of the 5th international hyperbaric conference, vol 2. British Columbia: Simon Fraser University 1974:691–698.
18. Lambertsen CF. Effects of excessive pressures of oxygen, nitrogen, helium, carbon dioxide and carbon monoxide. In: Mountcastle VB, ed. Medical physiology, vol 2, 14th ed. St. Louis, MO: C. V. Mosby, 1980:1901–1944.