

# The life-saving effect of hyperbaric oxygenation during early-phase severe blunt chest injuries.

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Rogatsky GG, Mayevsky A. The life-saving effect of hyperbaric oxygenation during early-phase severe blunt chest injuries. *Undersea Hyperb Med* 2007; 34(2):75-81. The effect of hyperbaric oxygenation (HBO<sub>2</sub>) on survival during the early phase of severe blunt chest injury (BChI) has not been elucidated. Our aim was to investigate this effect on human victims of BChI. We monitored cardiac index (CI), stroke volume index (SVI), PaO<sub>2</sub> and PaO<sub>2</sub>/FiO<sub>2</sub> in 18 victims treated conventionally, and 8 victims treated under combined conventional and HBO<sub>2</sub> treatment. Out of the 18 victims, 4 survived (Group A) and 14 died (Group B). Another 8 victims, in Group C, received HBO<sub>2</sub> and all survived. Human victims showed marked reductions in all cardiorespiratory values during the first 24 h. Group B persistently tended towards a decrease in SVI, PaO<sub>2</sub>/FiO<sub>2</sub> and PaO<sub>2</sub>, eventually reaching fatal levels. The survivors developed a cardiorespiratory function characterized by a tendency towards recovery of all monitored parameters, more notable in Group C, which showed an earlier and more significant normalization vs. Group A (P<0.01). Our clinical data suggest that the earliest possible HBO<sub>2</sub> treatment after severe blunt trauma can significantly enhance victims' survival.

## INTRODUCTION

Despite the extreme importance of rescue and therapy of severe blunt injury victims (including blast injuries), the role and efficiency of hyperbaric oxygenation (HBO<sub>2</sub>) in the management has not been investigated in detail. In this regard, perhaps the least studied are questions about the influence of HBO<sub>2</sub> treatment (HBO<sub>2</sub>T) on the victims' survival after severe blunt chest injuries (BChI). Previous studies showed that severe BChI is accompanied by early cardiorespiratory dysfunction (1-4). This dysfunction led to a drastic decrease in oxygen delivery, associated with poor outcome (2). Mortality increases to an even greater level upon BChI combined with injuries of other organs, specifically, with head injuries (HI), which often occur in the

most severe cases of blunt trauma (5,6).

This background indicates it is reasonable to evaluate the ability of HBO<sub>2</sub>T, the most powerful known anti-hypoxic strategy, to prevent life-threatening levels of tissue hypoxia at an early stage of severe blunt injury. The first such observations were performed over 30 years ago and showed that HBO<sub>2</sub>T had a beneficial effect on survival after severe BChI in animal experiments (7), and later in human victims (8). Since then, there has been a lack of contemporary data on the influence of early HBO<sub>2</sub>T on dynamics of cardiorespiratory function and, accordingly, on the survival of victims with a severe BChI. This study is a retrospective analysis of our clinical observations of severe BChI victims who underwent HBO<sub>2</sub>T in the course of their therapy.

## MATERIALS AND METHODS

The studies involving human subjects were performed according to the regulations of the institutional Helsinki Committee. The current report is based upon data obtained from 26 victims with severe BChI, treated in the ICU of Moscow Scientific Research Institute for Emergency Medicine (Dr. G.G. Rogatsky). All the victims suffered 4-12 rib fractures with contusion of the lungs. Pneumothorax was diagnosed in 10 victims and hemopneumothorax in 15 victims at the time of admission; two of the latter underwent surgery to control bleeding. Twenty victims were in a state of traumatic hemorrhagic shock at the accident site. Twenty-two of them also had injuries to other parts of the body (brain, abdomen, pelvis, and long bone), but BChI was the major factor in the victims' general condition.

Within the 24-72 h following blunt injury, all 26 victims developed arterial hypoxemia while receiving conventional treatment, and they comprise the cohort of the present study. The analysis involved dividing them into three groups according to outcome and therapy: Group A consisted of 4 survivors who were treated by conventional therapy only, Group B consisted of 14 victims who died after having been treated by conventional therapy only, and Group C consisted of 8 survivors who were treated by a combination of conventional and HBO<sub>2</sub> therapy.

Management at the time of admission consisted of measures for resuscitation of circulation and breathing. According to standard protocol, the victims were resuscitated with a transfusion of a solution of crystalloids and colloids, blood (or blood products) as indicated clinically, nasotracheal or endotracheal intubation as necessary, supplemental inspired oxygen, correction of acid-based changes in the blood, resolution of pneumo- and/or hemopneumothorax, necessary inotropic

support, and analgesia. Mechanical ventilation was used when needed for severe and resistant hypoxemia. All hyperbaric treatments were started during the first day after blunt injury and were performed in a monoplace chamber. The protocol for HBO<sub>2</sub> exposure was 1.6-2.0 ATA, 40-60 min daily for 4-15 consecutive days, and this was adjusted according to the progress of recovery.

Cardiac output index (CI) and stroke volume index (SVI) were measured by a noninvasive impedance cardiography technique (9,10) using corresponding standard formulas (11). Arterial blood gas values were measured as well (ABL-330, Radiometer). The measurement of all parameters, usually simultaneously, was carried out 1-3 times daily.

Statistical analysis was performed using Student's t-test. A value of  $p < 0.05$  was considered significant. Results are presented as mean  $\pm$  SD.

## RESULTS

The cardiorespiratory changes in victims of BChI were divided into three phases. The 1<sup>st</sup> phase, from the moment of trauma until up to 24 h, was marked by cardiorespiratory instability (sometimes with very severe changes in homeostasis) and recovery required intensive care. The 2<sup>nd</sup> phase, beginning on days 2-4 and continuing up to 26 days, was characterized by the appearance and development of acute cardiorespiratory dysfunction (ACRD). The 3<sup>rd</sup> phase lasted up to 2 days; this phase was set apart by rapid and fatal worsening of cardiorespiratory parameters among the non-survivors. In contrast, a relatively stable state to a near-normal level for cardiorespiratory parameters was attained among the surviving victims.

Table 1 presents the relevant features

**Table 1.** Characteristics of cardiorespiratory function in victims after severe blunt chest injury (mean ± SD).

Parameters	Phase	Type of therapy	Conventional therapy		Additional HBO <sub>2</sub> T	
		Survival	Survivors	Nonsurvivors	Survivors	Nonsurvivors
		No. of victims	N=14	N=14	N=8	N=0
		Group	A	B	C	
PaO <sub>2</sub> mm Hg	1		49.7±7.68	61.8±18.48	57.7±5.62	
	2		57.9±10.61	52.3±11.40	65.4±11.73*(1);	
	3		78.0±4.32 *** (1,2); †††(B)	40.1±7.62*** (1), ** (2)	††(B) 85.3±5.85 *(1,2); †††(B)	–
PaO <sub>2</sub> /FiO <sub>2</sub> ratio	1		248±38.38	308±91.08	290±27.91	
	2		284±53.33	255±53.97	323±57.65 *(1);	
	3		389±18.55 *** (1,2); †††(B)	173±33.86 *** (1,2)	††(B) 422±15.17 *** (1,2); †††(B)	–
SVI (ml m <sup>-2</sup> )	1		31.9±17.64	33.6±15.61	31.0±7.05	
	2		46.4±13.02	38.2±9.34	43.3±9.20** (1)	
	3		50.0±7.77 †(B)	27.3±5.57	47.7±2.79 ** (1); †††(B)	–
CI (l min <sup>-1</sup> )	1		3.77±2.42	3.14±0.97	3.16±0.71	
	2		4.76±1.19	4.66±1.20,* (1)	4.26±1.19,* (1)	
	3		4.34±0.87	3.42±1.23	4.17±1.22	–
HR (b min <sup>-1</sup> )	1		108±25.85	98±22.52	99±14.10	
	2		101±12.57†(B)	132±28.79* (1)	94±15.54†	
	3		90±10.78 †(B)	116±22.02	82±13.45 *(1); ††(B)	–

PaO<sub>2</sub> = arterial partial pressure of oxygen; PaO<sub>2</sub>/FiO<sub>2</sub> ratio = ratio of the partial pressure of arterial oxygen to the fraction of inspired 100% oxygen.

SVI = stroke volume index; CI = cardiac index; HR = heart rate. See text for definitions of phases 1-3.

This table shows statistically significant differences of mean values after comparison of all phases and groups. Values are shown as mean (SD). \*Significant differences between phases, P<0.05; \*\*Significant difference between phases, P<0.01; \*\*\*Significant difference between phases, P<0.001. The number inside the brackets beside symbols represents phase number. †Significant difference between Groups A (or C) and B, P<0.05; †Significant difference between Groups A (or C) and B, P<0.01; †††Significant difference between Groups A (or C) and B, P<0.001.

of the data. In all groups, the 1<sup>st</sup> phase showed a profound reduction in the mean values for the cardiorespiratory parameters (PaO<sub>2</sub>/FiO<sub>2</sub> ratio, PaO<sub>2</sub>, SVI, CI) and tachycardia. The 2<sup>nd</sup> phase was characterized by a tendency towards recovery of these parameters in Groups A and C, but not in Group B. This tendency was more pronounced in Group C, for which a statistically

significant increase of the mean values of these parameters was already apparent in the 2<sup>nd</sup> phase compared with the 1<sup>st</sup> phase. The 3<sup>rd</sup> phase in Groups A and C was characterized by recovery of these parameters to normal or near-normal levels. As a result, mean values of the parameters in the 3<sup>rd</sup> phase were significantly higher than those in the 1<sup>st</sup> phase (PaO<sub>2</sub>/FiO<sub>2</sub> ratio, PaO<sub>2</sub>,

SVI) and even the 2<sup>nd</sup> phase (PaO<sub>2</sub>/FiO<sub>2</sub> ratio, PaO<sub>2</sub>). Despite the trends, normalization of the mean values of the measured cardiorespiratory parameters were observed in the 3 phases only in Group C.

There were different qualitative changes in Group B. After the initial reduction of parameters in the 1<sup>st</sup> phase, subsequent worsening cardiorespiratory function was observed in the 2<sup>nd</sup> and 3<sup>rd</sup> phases. In fact, the reduced means of the PaO<sub>2</sub> and PaO<sub>2</sub>/FiO<sub>2</sub> ratio levels – significantly lower in the 3<sup>rd</sup> phase than in the 1<sup>st</sup> and 2<sup>nd</sup> phases – yielded a critically lower mean SVI. Compared to the 1<sup>st</sup> phase, CI increased only for a while in the 2<sup>nd</sup> phase due to tachycardia. Extremely low levels of PaO<sub>2</sub> and SVI in the 3<sup>rd</sup> phase were ultimately fatal for these victims.

As a result of the combination of tendencies above, the differences in mean SVI, PaO<sub>2</sub> and PaO<sub>2</sub>/FiO<sub>2</sub> ratio in Group A and even more so in Group C took on increasing importance compared with the same parameters in Group B, becoming maximal in the 3<sup>rd</sup> phase ( $P < 0.001$ ). The mortality rate in the two groups with conventional therapy (Groups A + B) was 77%, and 0% in Group C.

## DISCUSSION

Analysis of data presented in Table 1 indicates that with the dramatic decrease in mean PaO<sub>2</sub>/FiO<sub>2</sub> ratio characteristic for all groups studied, the second phase can be considered the onset of fatal decline in this parameter, when the tendency of decline was already more pronounced than in the other groups (255±53 vs 284±53 and 323±57 in groups B, A, and C, respectively).

This level of PaO<sub>2</sub>/FiO<sub>2</sub> the non-surviving victims in the second phase, agrees with the conclusions of recent investigations that point to a risk of lethal outcome for

victims of acute lung injury (ALI) and ARDS when the PaO<sub>2</sub>/FiO<sub>2</sub> ratio reaches 300 mmHg (12,13) and even 350 mmHg (14,15). These levels are much higher than the level of 250 mmHg posited in earlier works (16,17, and others). In addition, there is a basis for assuming that when assessing the probability of lethal outcome under such conditions, it is important (perhaps even essential) to consider not only the absolute PaO<sub>2</sub>/FiO<sub>2</sub> ratio, but the direction and dynamics of its changes. In our study, the use of HBO<sub>2</sub>T qualitatively stalled the negative trends in this parameter, starting with the second phase. This is evidenced by the statistically significant differences in the PaO<sub>2</sub>/FiO<sub>2</sub> ratio in the second and third phases in group C, with a corresponding level in group A (i.e., the group of survivors that received conventional treatment).

In our study, the PaO<sub>2</sub>/FiO<sub>2</sub> ratio proved to be a highly adequate and informative parameter for assessing the severity of acute respiratory dysfunction and therapy efficiency. It is important to note that a recent study using large groups (18), showed that the PaO<sub>2</sub>/FiO<sub>2</sub> ratio is a simple method of quantifying lung injury severity in trauma patients that better predicts mortality compared with the more complex modified Murray lung injury score. According to these authors, “the P/F score should replace more complex and potentially therapy-dependent scores” (18). Our observations agree with these conclusions, especially concerning the functional utility of this parameter in pathophysiologic analysis of the victims’ cardiorespiratory status.

The data presented in Table 1 indicate the development of acute cardiorespiratory dysfunction (ACRD) after blunt injury is characterized by reduction not only of pulmonary gas exchange but also of cardiac function. Taken together with the PaO<sub>2</sub>/FiO<sub>2</sub> ratio and the PaO<sub>2</sub> value, the SVI is a highly prominent marker, especially in the group

of victims with a fatal outcome for whom intensive therapy failed to restore and stabilize cardiorespiratory function.

In analyzing the clinical course in Group C, it appears that cardiopulmonary resuscitation in victims with poor cardiac function who were treated with HBO<sub>2</sub> was similar to that of Group A. Moreover, the statistically significant increase of PaO<sub>2</sub>/FiO<sub>2</sub>, PaO<sub>2</sub>, and SVI that was already apparent in the 2<sup>nd</sup> phase relative to the 1<sup>st</sup> phase, suggests that reversibility in Group C was more apparent than in Group A. As a result, victims treated with a combination of conventional and HBO<sub>2</sub> therapy in the 3<sup>rd</sup> phase can be expected to normalize their cardiorespiratory parameters. It is also noteworthy that the absence of mortality in 8 victims treated with HBO<sub>2</sub> may indicate that HBO<sub>2</sub> is especially suitable for victims with poor cardiac function following BChI. The positive effect of adjunctive HBO<sub>2</sub> on these victims may include powerful anti-hypoxic potential that is capable of effective correction of disorders induced by acute deficit of oxygen to tissues (19-22). These conclusions are also supported by our published results in animals (23,24). It was established that the application of HBO<sub>2</sub> during the early phase of severe head injury significantly diminished intracranial pressure and mortality (25).

The implementation of HBO<sub>2</sub> as considered above can be linked therapeutically by the delivery of greatly-needed oxygen to the tissue with subsequent prolonged improvement in metabolism of the traumatized brain, as shown by Rockswold et al. (26). The earliest possible HBO<sub>2</sub> application (during emergency care), while correcting the oxygen deficiency caused by the acute decrease in cerebral blood flow and acute respiratory dysfunctions (27-29), as well as activating tissue metabolism, might provide for the early stabilization of the victim's state.

Another mechanism responsible for the

pathogenesis of critical conditions resulting from a severe blunt injury should also be noted. Studies of the past have established that the development of acute cardiopulmonary dysfunction is already present at an initial stage of severe head trauma (30-33). In this case, cardiopulmonary dysfunction induces pronounced pathophysiological perturbations, usually accompanying severe HI (such as apnea, hypoxia, hypercarbia, and catecholamine surge (34). Excessively high catecholamine levels cause myocardial damage and cardiac failure (34,35). A markedly low cardiac index may result from the heart failure (1,30,32). Therefore, under these conditions as well as in cases of BChI, the application of HBO<sub>2</sub> can reinforce the resistance to ACRD. Accordingly, elimination of progressive arterial and tissue hypoxia relieves a "physiologic depressant" of the heart (36) through the use of HBO<sub>2</sub>, and it is possible to prevent, or at least delay, acute progressive disturbances in myocardial contractility.

This conclusion appears to be supported by data showing restoration of hypoxic myocardial contractions after treatment with HBO<sub>2</sub> in humans (37,38) and even an increase in cardiac contractility in healthy animals (39,40). Thus, the significant recovery of SVI following HBO<sub>2</sub> administration may create a potential for normalization not only of CI levels, but also of lung gas exchange by this approach to therapy for ACRD. We suppose that cardiac function is a determining factor in victims who have undergone severe blunt trauma. Elimination of the cardiac component of hypoxia in these victims was probably no less important than elimination of the "pure" pulmonary component, because restoration of the necessary level of SVI and CI effectively solved the problem of inadequate oxygen delivery to tissues.

As can be seen from Table 1, the recovery of respiratory function in survivors

(Groups A and C) was seen most clearly by the tendency for SVI to increase in the 2<sup>nd</sup> and 3<sup>rd</sup> phases. We propose that adequate therapy interrupts the “vicious circle” of interconnected acute deteriorations of respiratory and cardiac functions, and that the main goal of therapy should be the elimination of hypoxic and circulatory hypoxia. Therefore, HBO<sub>2</sub> should be considered a front-line treatment with a demonstrated capability to improve cardiorespiratory function.

In conclusion, the early-phase application of HBO<sub>2</sub>T in severe BChT (under emergency care regimen) dramatically lowers the mortality. Analysis of our results in comparison to the data in the literature, justifiably suggests extrapolation of these ideas to victims of primary blast injury.

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