CORRECTION OF AN EXPERIMENTAL ACUTE RESPIRATORY FAILURE SYNDROME BY HYPERBARIC OXYGENATION

G. G. Rogatskii, M. B. Vainshtein, and T. V. Sevost'yanova

UDC 616.24-008.64-06:616.152.. 21-008.64-085.835.12

KEY WORDS: arterial hypoxemia; hyperbaric oxygenation.

Despite the unenthusiastic or definitely negative attitude of many investigators to the idea of using high oxygen concentrations in acute lung lesions [2, 5, 6, 11, 12], several reports have been published in recent years by authors who have noted the beneficial effect of hyperbaric oxygenation (HBO) as a constituent of the treatment of patients with acute respiratory (pulmonary) failure [1, 4, 7] and also in severe experimental contusions of the lungs [10]. Nevertheless, in an analysis of data in the literature, we could find no information on special investigations into the possible use of HBO to correct the acute respiratory failure syndrome (ARFS) or on the mechanisms of its actions under these circumstances.

The aim of this investigation was to study the effect of HBO on the course of experimental ARFS.

EXPERIMENTAL METHODS

Experiments were carried out on 159 Wistar rats weighing 150-350 g. In the experiments of series I (control) a model of ARFS was produced in 54 animals by a single injection of oleic acid (OA) into the right pleural cavity in a dose of 0.3 ml/100 g body weight. Animals of series II (34 rats) and III (51 rats) underwent one session of HBO with a pressure of 2 atm for 2 h and 4 atm for 12 h, respectively, after the injection of OA, and the animals of group IV (five rats) were subjected to HBO with a pressure of 2 atm for 4 h. Changes in metabolic activity of the animals under the influence of these procedures were assessed by means of one of the most informative parameters of the pulmonary gas exchange, namely the partial pressure of oxygen in arterial blood (p_aO_2), and also on the basis of macroscopic assessment of changes in the lungs observed during thoracotomy, and the level of mobility of the animals. Blood samples were obtained by puncture of the femoral artery or abdominal aorta under open ether anesthesia. For the morphological control the animals were decapitated immediately after blood sampling. Values of p_aO_2 were measured by the "Corning M-165" blood gas microanalyzer (England). The state of the pulmonary gas exchange and the structure of the lung tissue in series I were studied before injection of OA and 4, 12, 18, and 24 h after its injection, and in series II and III, 1, 12, 24, and 48 h and 8-15 days after exposure to HBO. The numerical results were subjected to statistical analysis.

EXPERIMENTAL RESULTS

Injection of OA caused the development of a pathological state accompanied by a progressive decrease in p_aO_2 (Fig. 1). The severity and rapidity of development of the process were such that most animals of the control series (80%) died between 12 and 24 h after the injection of OA, and 10% of the rats died during the next 2 days (Table 1). Inspection of the lungs of the animals dying in the early stages revealed hyperemia and foci of hemorrhage, while in the later stages, other changes included atelectasis, which became subtotal or total in both lungs by the end of the period of observation (usually more severe on the right), and it led to "hepatization" of the lungs. Fragments of lung excised from virtually anywhere sank in water. In some cases a frothy fluid was present in the lumen of the trachea and bronchi, evidence of pulmonary edema.

Laboratory of Experimental Pathology, N. V. Sklifosovskii Emergency Aid Research Institute, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR A. P. Ado.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 105, No. 4, pp. 410-411, April, 1988. Original article submitted May 22, 1987.

0007-4888/88/0004-0485$12.50

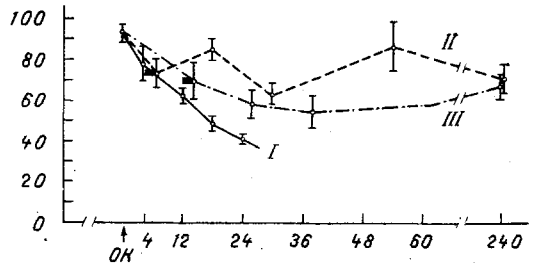

Fig. 1. Dynamics of p_aO_2 after intrapleural injection of OA (0.3 ml/100 g). Abscissa, time (in h); ordinate, p_aO_2 (in mm Hg). I) Control series; II, III) series of experiments with HBO, carried out 4 and 12 h, respectively after injection of LA. Shaded rectangles indicate period of HBO session. Arrow indicates time of injection of OA.

Table 1. Mortality (in %) of Animals after Intrapleural Injection of OA (0.3 ml/100 g)

Period of observation after injection of OA	Series I (control)	Series II and III (main group)
24 h	80	2
72 h	90	10
30 days	96	19

A single session of HBO caused a marked change in the character of the pathological process induced by injection of OA. The main feature of this change was a marked fall of mortality (Table 1). In the animals of the main group (series II and III) those rats which died did so later than in the control. On the whole, it can be concluded from analysis of the results that the viability of the rats in the main group at the most critical period for them, namely the first 3 days, was increased by several times, and this was accompanied also by a marked increase in their mean life span at each time of observation.

Analysis of the values of p_aO_2 showed that in the rats of the experiments of series II and III the degree of arterial hypoexmia was less than in the control. The decrease of this parameter as a rule was reversible in character. Even 12 h after injection of OA (i.e., at a time toward the end of which some of the animals in series I died) performance of an HBO session led to initial stabilization of the average p_aO_2 level at close to the limiting value, followed by a gradual rise (although this was not significant) in the value of the parameter. In series II, however, the time course of the average p_aO_2 level was more favorable still: minimal values were not below 60 mm Hg, even at the most critical period of observation for the animals' life. At later stages (after 10-15 days) the level of arterial hypoxemia was not threatening.

Changes in the lungs found at autopsy on the rats in the early stages after the HBO session consisted of local atelectases or small hemorrhages, mainly subpleural in their distribution. The lungs were mainly filled with air (except in the areas of atelectasis). In the later stages adhesions formed in the pleural cavity, with appreciable signs of deformation of the lungs and pericardium.

Changes found in the lungs of animals dying after HBO were virtually indistinguishable from those in the control.

The use of an HBO session in the experiments of series IV did not prevent death of the animals. Indeed, four of five rats died during the first 48 h after the session. The clinical and morphological picture in these experiments was the same as in series I.

It can be tentatively suggested that the definitely positive results of the HBO session in series II and III is due primarily to the fact that the conditions of hyperbaric oxygenation chosen were appropriate. The observations show that keeping the animals for longer in the pressure chamber (4 h) under standard conditions of excess oxygen pressure did not give a favorable effect. These observations are in agreement with the results of an earlier

study of the effect of HBO on intact lung tissue [8]. The schedule and duration of HBO chosen evidently created conditions under the circumstances of the investigation which, on the one hand, maintained the necessary level of activation of metabolism in the lung tissue [3, 9] and, on the other hand, were insufficient to give rise to toxic lung damage by oxygen. The stimulus obtained as a result of this procedure evidently facilitates the formation of a stable defensive and adaptive response at different levels, including the whole organ (lung), even in the presence of severe diffuse involvement of this tissue. Our results, confirm the previous concept, according to which HBO has an adaptive effect also in acute respiratory failure due to the development of the so-called "wet lung."

LITERATURE CITED

1. A. B. Aksel'rod, in: A Manual of Hyperbaric Oxygenation [in Russian], ed. S. N. Efuni, Moscow (1985), pp. 197-201.
2. A. P. Zil'ber, Artificial Ventrilation of the Lungs in Acute Respiratory Failure [in Russian], Moscow (1978).
3. A. N. Leonov, Hyperbaric Medicine [in Russian], Vol. 2, Moscow (1983), pp. 7-9.
4. B. K. Namazbekov, "Hyperbaric oxygenation in the prevention and combined treatment of suppurative complications of operations on the lungs and pleura," Dissertation for the Degree of Candidate of Medical Sciences, Moscow (1986).
5. N. K. Permyakov, The Pathology of Resuscitation and Intensive Care [in Russian], Moscow (1985).
6. "Indications for and contraindications to hyperbaric oxygenation," in: A Manual of Hyperbaric Oxygenation [in Russian], ed. S. N. Efuni, Moscow (1985), pp. 390-393.
7. G. G. Rogatskii, Yu. V. Isakov, E. A. Pisarenko, and V. A. Strokov, Anest. Reanimatol., No. 3, 60 (1980).
8. G. G. Rogatskii, P. P. Golikov, M. A. Sapozhnikova, et al., Patol. Fiziol., No. 1, 56 (1987).
9. L. L. Shimkevich, Hyperbaric Medicine [in Russian], Vol. 2, Moscow (1983), pp. 45-47.
10. E. G. Damon and R. K. Jones, Physiologist, 14, No. 3, 127 (1971).
11. K. J. Dobuler, J. D. Catravas, and N. Gillis, Am. Rev. Resp. Dis., 126, No. 3, 534 (1982).

PHARMACOLOGIC ANALYSIS OF CHANGES IN MORPHOLOGY AND FUNCTION OF ADRENERGIC STRUCTURES OF THE PANCREAS IN LESIONS OF ITS ENDOCRINE AND EXOCRINE DIVISIONS

T. N. Nilova, R. V. Vashetko, and M. D. Khegai

UDC 616.379-008.6-02:616.839.1

KEY WORDS: pancreas; adrenergic structures; pharmacologic analysis.

The role of the sympathetic nervous system (SNS) in the regulation of the exocrine and endocrine functions of the pancreas is generally accepted. However, its role in the development of the various pathological processes and their pharmacologic correction by sympathicotropic agents have been studied very inadequately.

The aim of this investigation was to undertake a combined morphologic, biochemical, and pharmacologic investigation of the adrenergic components of the autonomic innervation of the pancreas in various types of experimental pathology of its exocrine and endocrine divisions.

Department of Pharmacology, Research Institute of Experimental Medicine, Academy of Medical Sciences of the USSR. Laboratory of Pathmorphology, I. I. Dzhanelidze Emergency Aid Research Institute, Leningrad. Department of Pathological Physiology, Leningrad Sanitation and Hygiene Medical Institute.

(Presented by Academician of the Academy of Medical Sciences of the USSR B. I. Tkachenko.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 105, No. 4, pp. 412-414, April, 1988. Original article submitted December 18, 1986.

0007-4888/88/0004-0487$12.50