

Rapid Recovery Hyperbarics

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Lung Disorders

Experimental

Hyperbaric oxygen attenuates lipopolysaccharide-induced acute lung injury M.-Y. Lu¹, B.-H. Kang¹, F.-J. Wan¹, C.-S. Chen² and K.-L. Huang^{1, 2},

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Abstract

Objectives. To study the effect of hyperbaric oxygen therapy in alleviating acute lung injury induced by lipopolysaccharide (LPS) in rats.

Design and interventions. The rats received an intraperitoneal injection of LPS (15 mg/kg). Animals were either breathing air at 1 ATA or subjected to hyperbaric oxygen (HBO₂) therapy. The HBO₂ therapy was carried out in a hyperbaric chamber at a pressure of 3 ATA for 90 min. In another two groups, LPS-treated rats also received intraperitoneal injection of N-nitro-L-arginine (L-NAME, 25 mg/kg) or L-N⁶-(iminoethyl)lysine (L-NIL, 10 ml/kg). Another two groups of LPS-treated rats were subjected to HBO₂ exposure after the injection of L-NAME or L-NIL.

Measurements and main results. The bronchoalveolar lavage (BAL) was done into the left lung at 7.5 h after intraperitoneal injection of LPS. Parts of the right lung were excised for myeloperoxidase measurement, whereas the rest was collected for wet/dry ratio determination. LPS significantly increased the nitrite/nitrate (NO_x⁻) concentration (34.4±15.7 vs 4.5±3.1 μM), LDH activity (66±17 vs 46±15 mAbs/min), and protein concentration (373±119 vs 180±90 mg/l) in the BAL fluid. Treatment with HBO₂ immediately after the injection of LPS enhanced the increase of NO_x⁻ production, but reduced the LDH and protein in BAL fluid to the control levels. Pretreatment with either L-NAME or L-NIL abolished the increase of NO_x⁻ in the BAL fluid and further elevated the LDH level and protein concentration.

Conclusion. Our results suggested that HBO₂ alleviates the LPS-induced acute lung injury, which may be related to the enhancement of nitric oxide production.

Keywords. Lipopolysaccharide - Acute lung injury - Hyperbaric oxygen - Nitric oxide

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