Hyperoxic and Hyperbaric-Induced Cardioprotection: The Role of Nitric Oxide Synthase3

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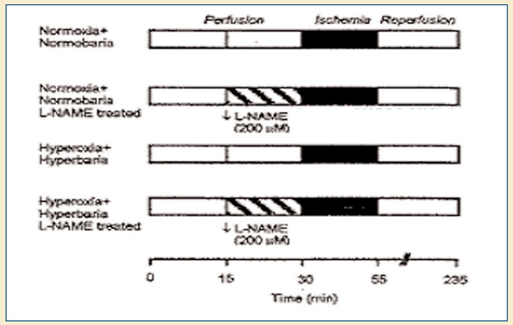
Abstract

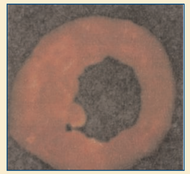
The relative contributions of atmospheric pressure (ATM) and F_IO_2 to cardioprotection are unknown. We determined whether the product of F_IO_2 x ATM (oxygen partial pressure) controls the extent of hyperbaric + hyproxic-induced cardioprotection and involves activation of nitric oxide synthase (NOS). Adult Sprague Dawley rats (n=10/gp) were treated for 1 hour with (1) normoxia + normobaria (21% 0_2 at 1 ATM), (2) hyperoxia + normobaria (100% 0_2 at 1 ATM) and (3) hyperoxia + hyperbaria (100% 0_2 at 2 ATM). Infarct size following 25 min ischemia and 180 min reperfusion was decreased following hyperoxia + hyperbaria treatment. L-NAME (200µM) a general NOS inhibitor, reversed the cardioprotective effects of hyperoxia + hyperbaria. Nitrite plus nitrate content was increased 2.2 fold in rats treated with hyperoxia + hyperbaria. NOS3 protein increased 1.2 fold and association of hsp90 with NOS3 4 fold in hyperoxia + hyperbaria rats. Cardioprotection conferred by hyperoxia + hyperbaria is directly dependent on oxygen availability and mediated by NOS.



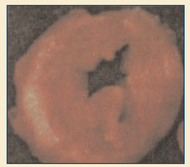
Methods of Cardioprotection

Infarct Size

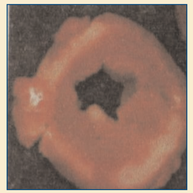
Hyperoxia + Hyperbaria
2 ATA, 100% FiO_2



Hyperoxia + Normobaria
1 ATA, 100% FiO_2

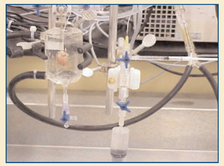


Normoxia + Normobaria
1 ATA, 21% FiO_2

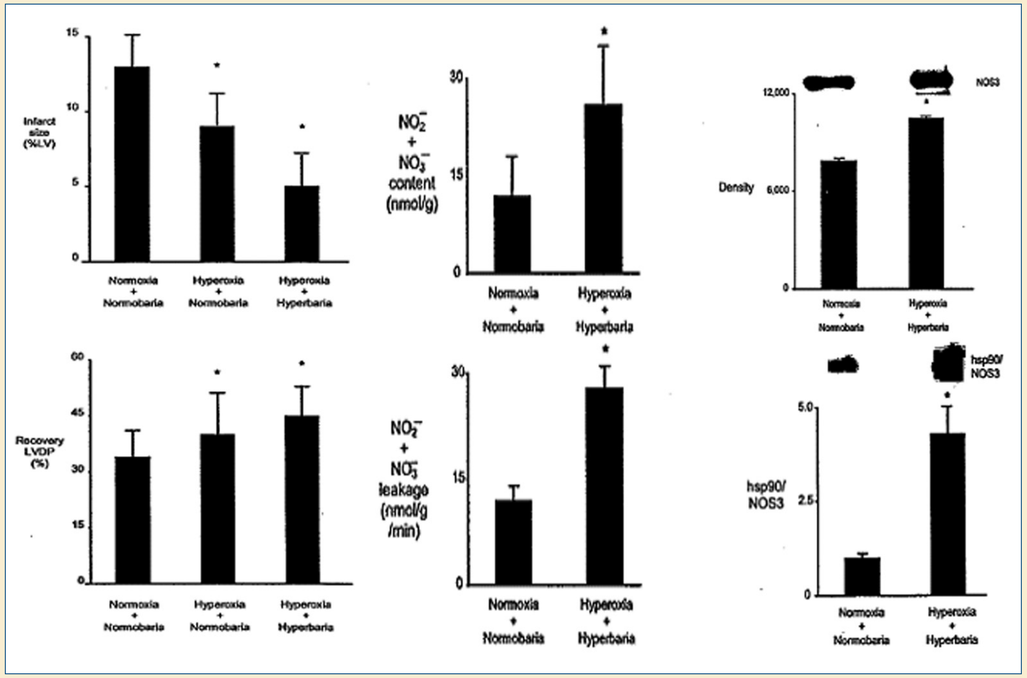


Sprague Dawley
8 week old rats

HBO Chamber



Langendorff Heart
Perfusion System



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