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## **Attenuating brain inflammation, ischemia, and oxidative damage by hyperbaric oxygen in diabetic rats after heat stroke.**

[Lee KL](#), [Niu KC](#), [Lin MT](#), [Niu CS](#).

### **Source**

Department of Surgery, Chi Mei Medical Center, Tainan, Taiwan.

### **Abstract**

#### **BACKGROUND/PURPOSE:**

Alternating hypothalamic-pituitary-adrenal axis mechanisms would lead to multiple organs dysfunction or failure. Herein, we attempt to assess whether hypothalamic inflammation and ischemic and oxidative damage that occurred during heatstroke (HS) can be affected by hyperbaric oxygen (HBO2) therapy in streptozotocin-induced diabetic rats.

#### **METHODS:**

In this study, anesthetized diabetic rats, immediately after the onset of HS, were divided into two major groups and given the normobaric air (21% O<sub>2</sub> at 1.0 atmospheres absolute) or HBO<sub>2</sub> (100% O<sub>2</sub> at 2.0 atmospheres absolute). HS was induced by exposing the animals to heat stress (43°C). Another group of anesthetized diabetic rats was kept at normothermic state and used as controls.

#### **RESULTS:**

The survival time values for the HBO<sub>2</sub>-treated HS-diabetic rats increased from the control values of 78-82 minutes to new values of 184-208 minutes. HBO<sub>2</sub> therapy caused a reduction of HS-induced cellular ischemia (e.g., increased cellular levels of glutamate and lactate/pyruvate ratio), hypoxia (e.g., decreased cellular levels of PO<sub>2</sub>), inflammation (e.g., increased cellular levels of interleukin-1 $\beta$ , tumor necrosis factor-alpha, interleukin-6, and myeloperoxidase), and oxidative damage (e.g., increased values of nitric oxide, 2,3-dihydroxybenzoic acid, glycerol, and neuronal damage score) in the hypothalamus of the diabetic rats.

#### **CONCLUSION:**

Our results suggest that, in diabetic animals, HBO<sub>2</sub> therapy may improve outcomes of HS in part by reducing heat-induced activated inflammation and ischemic and oxidative damage in the hypothalamus and other brain regions.

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#### **KEYWORDS:**

heatstroke, hypothalamus, inflammation, ischemia, oxidative damage

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