



HBO₂-Triggered Baroreflex: Mechanisms, Pathways, Benefits, and Limitations

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Background

Hyperbaric hyperoxia causes cardiovascular responses (vasoconstriction, bradycardia, cardiac output reduction) and shifts autonomic balance by increasing parasympathetic tone and decreasing sympathetic drive. We have hypothesized that these physiological responses are coordinated through the HBO₂-activated arterial baroreflex.

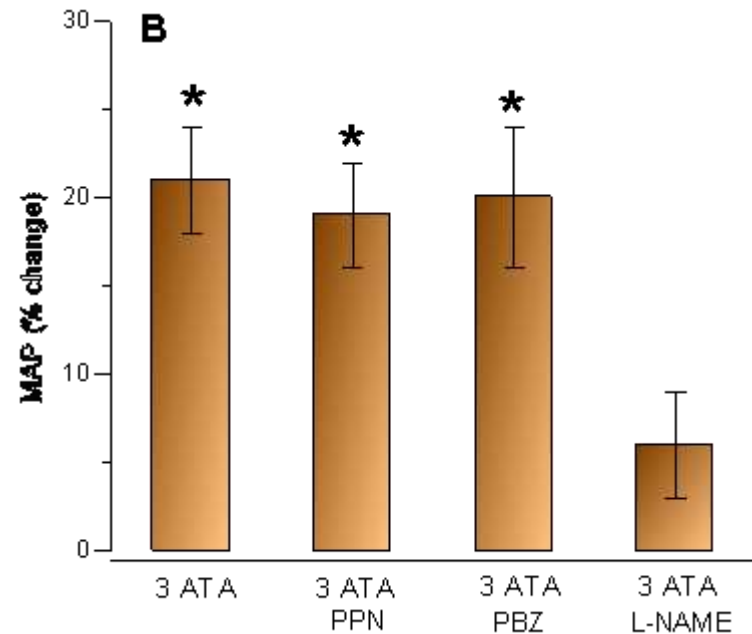
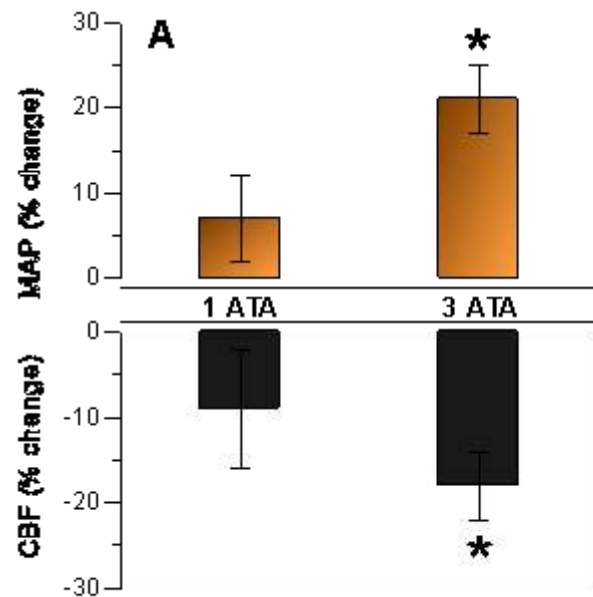
Aims

To evaluate mechanisms of baroreflex initiation in HBO₂, its effects on cardiovascular function and brain excitability, its functional limitations and contribution to HBO treatment.

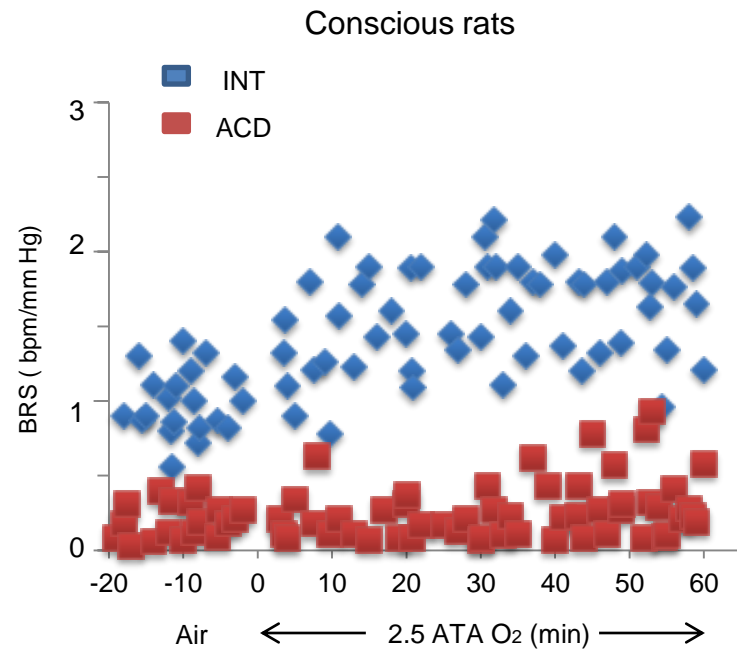
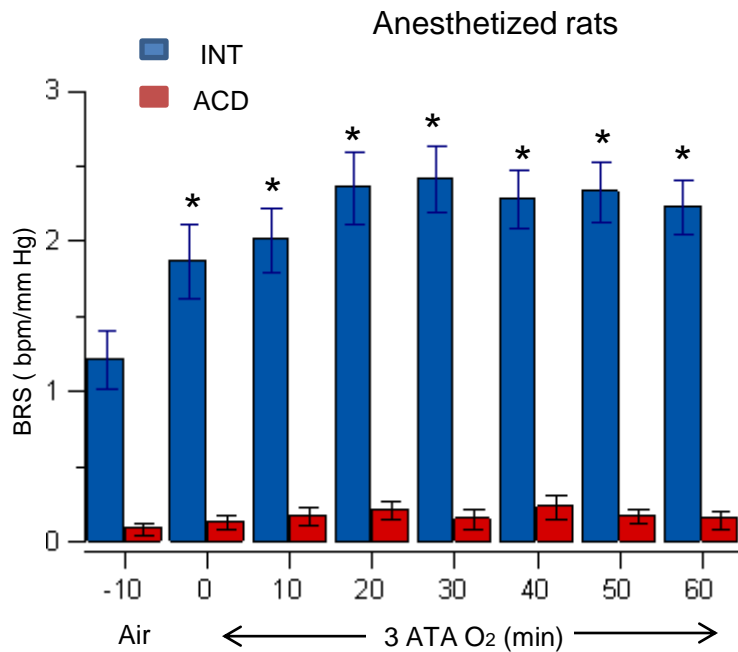
Methods and Approaches

<i>Animals:</i>	Anesthetized and conscious SD rats
<i>HBO₂:</i>	2.5 - 6 ATA
<i>Measurements:</i>	Arterial and ventricular pressures, cardiac output, heart rate, cerebral blood flow, total protein in BAL fluid
<i>Monitoring:</i>	EEG, ECG, renal sympathetic nerve activity (RSNA) and body temperature
<i>Calculations:</i>	Systemic and cerebral vascular resistance, baroreflex sensitivity (BRS)
<i>Interventions:</i>	Aortic and carotid baroreceptor deafferentation, electrical stimulation of aortic depressor nerve (ADN)

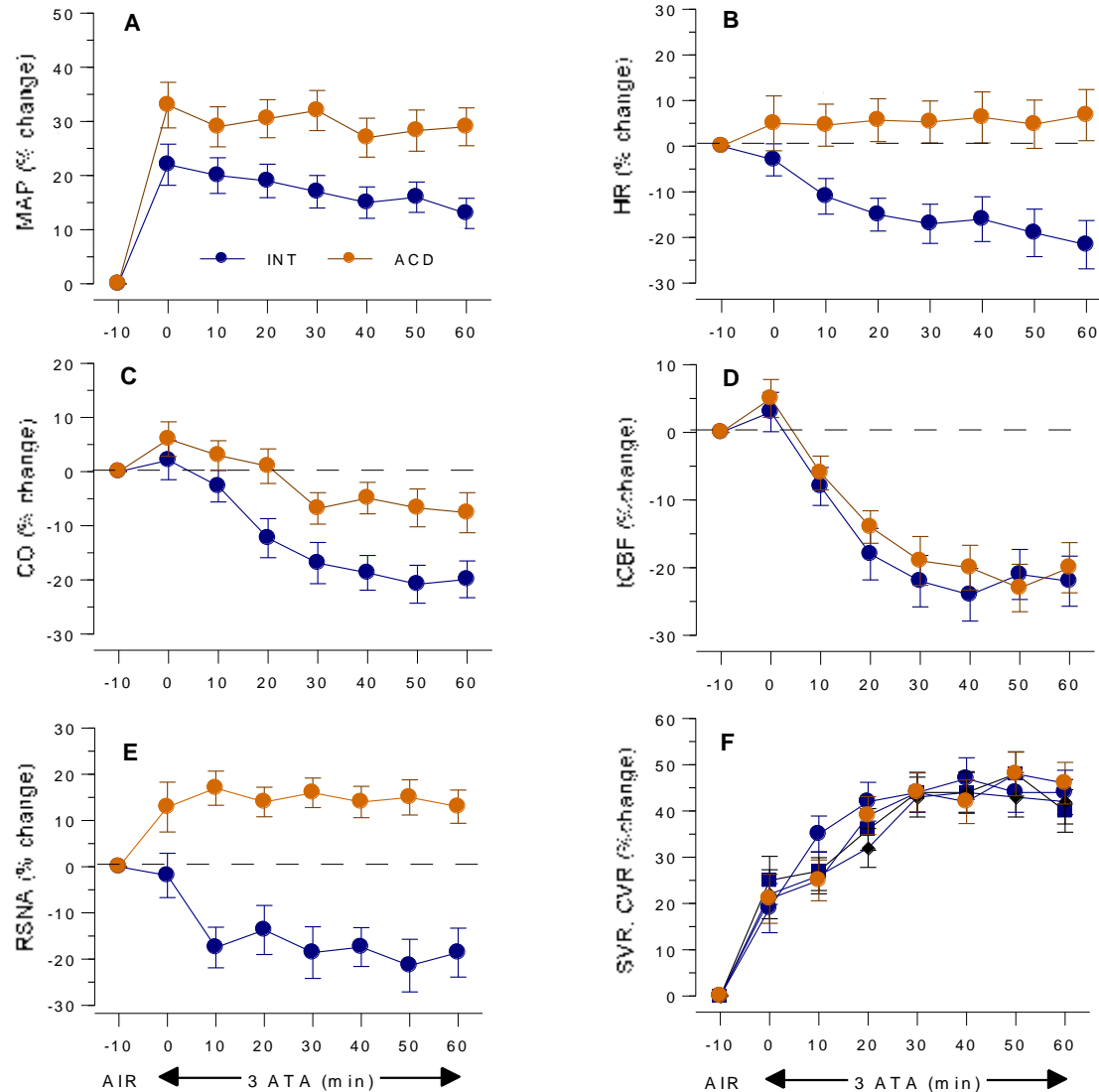
HBO₂ Triggers Baroreflex by Increase in Blood Pressure Due to Peripheral Vasoconstriction from Reduced Nitric Oxide Bioavailability



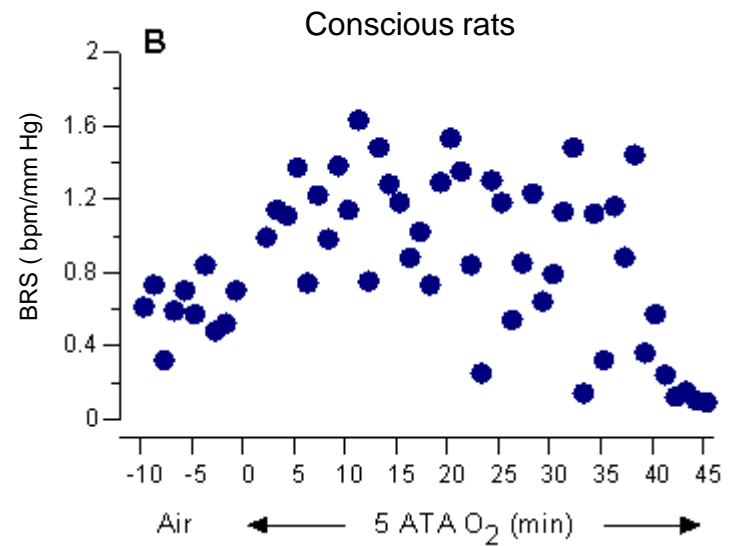
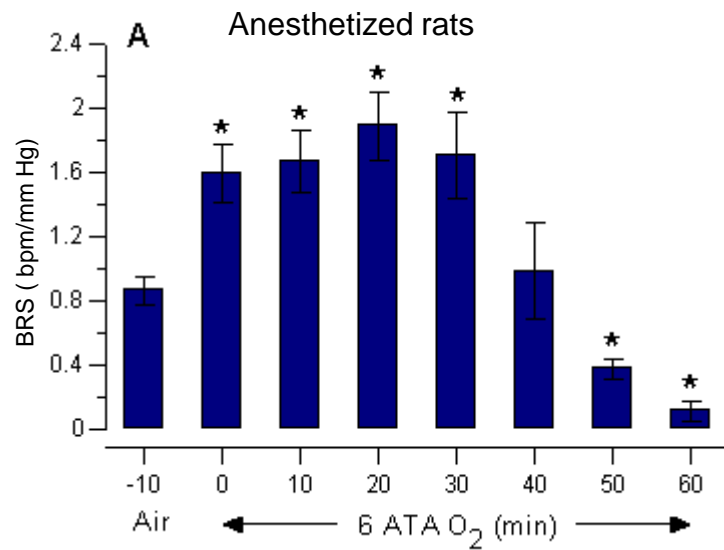
Moderate Hyperbaric Hyperoxia Improves Baroreflex Function by Activation of Baroreceptor Afferents



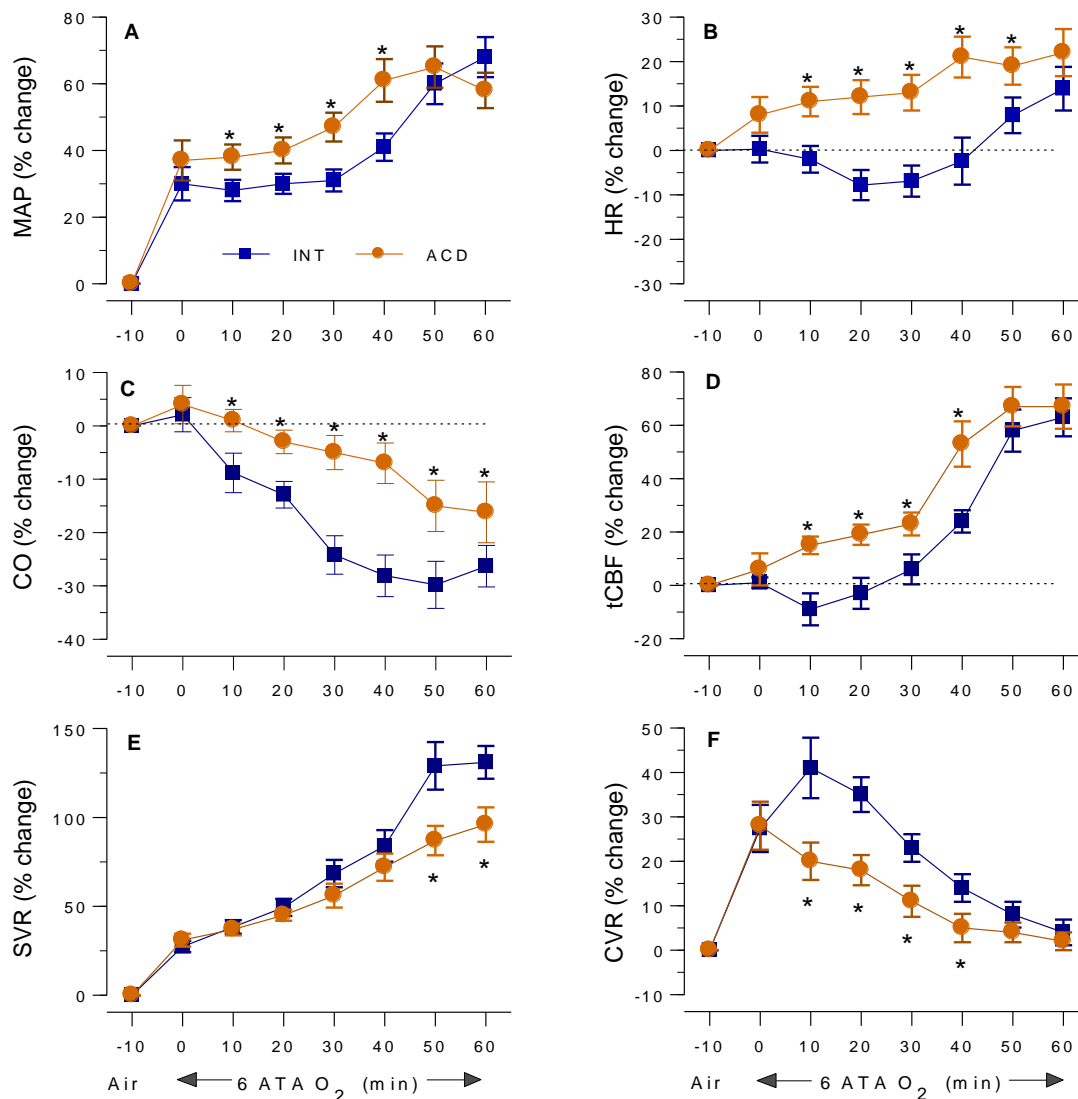
Effect of Baroreceptor Deafferentation on Cardiovascular and Autonomic Responses to HBO₂



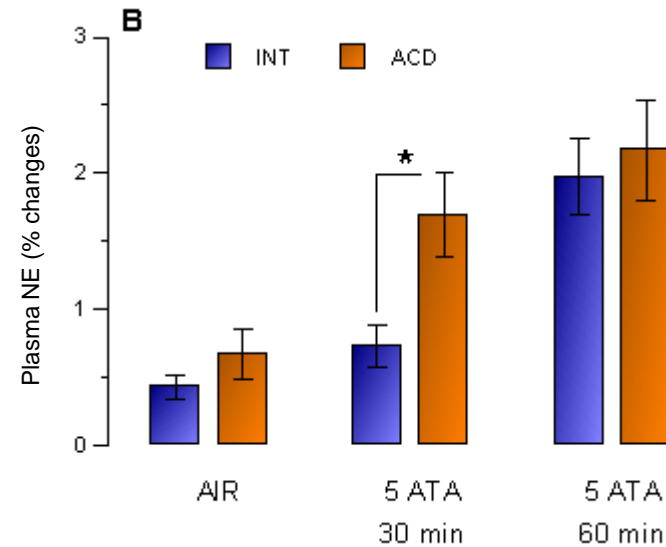
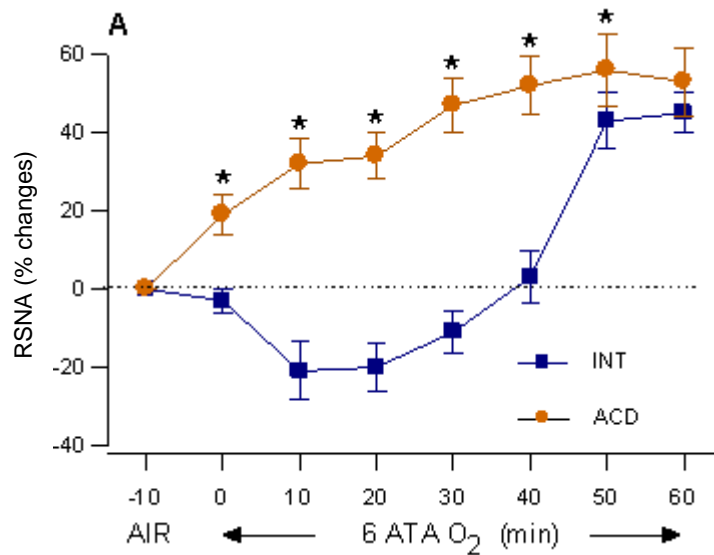
Baroreflex Function is Impaired in Extreme HBO₂



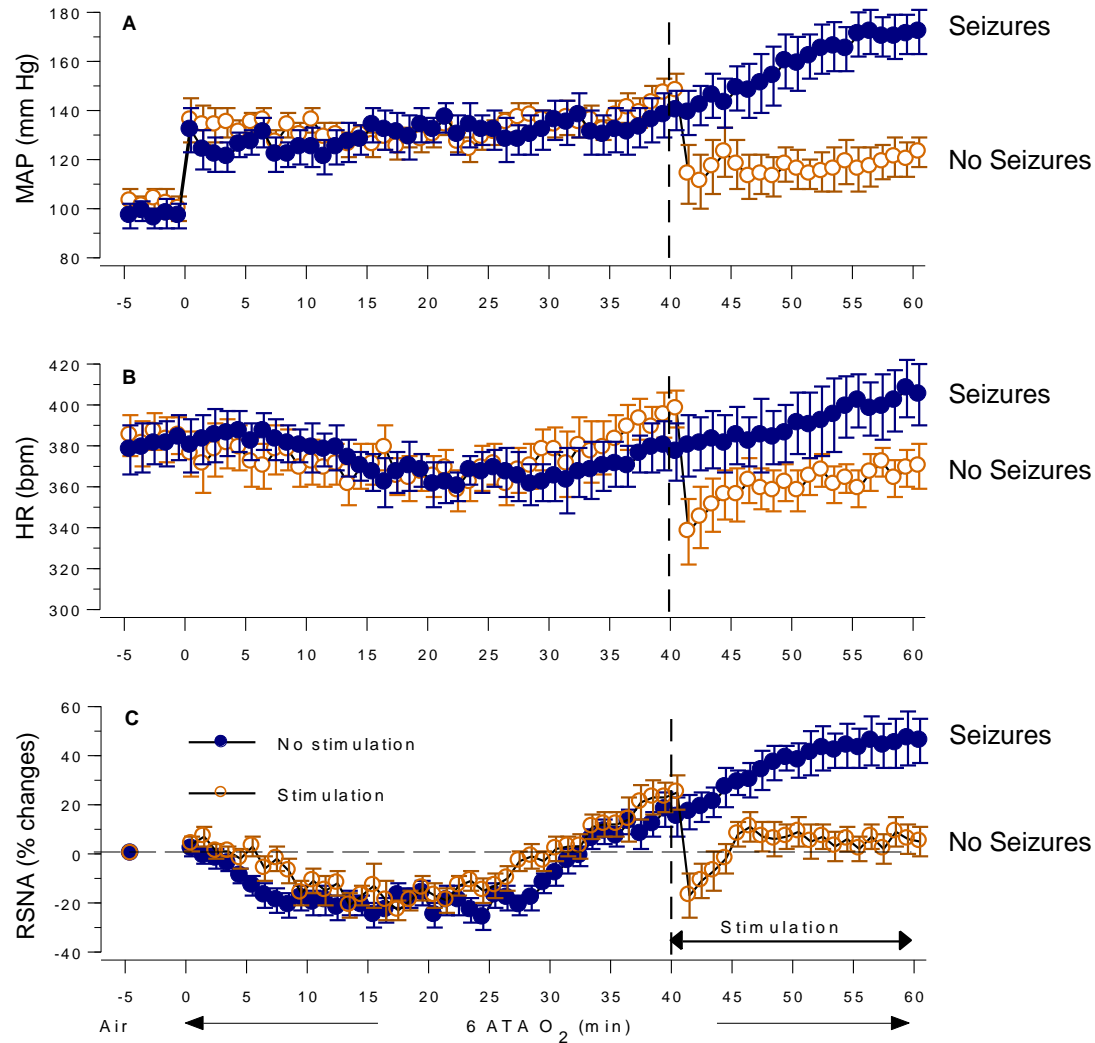
Baroreflex Impairment is Associated with Disturbances of Cardiovascular Functions in Extreme HBO₂



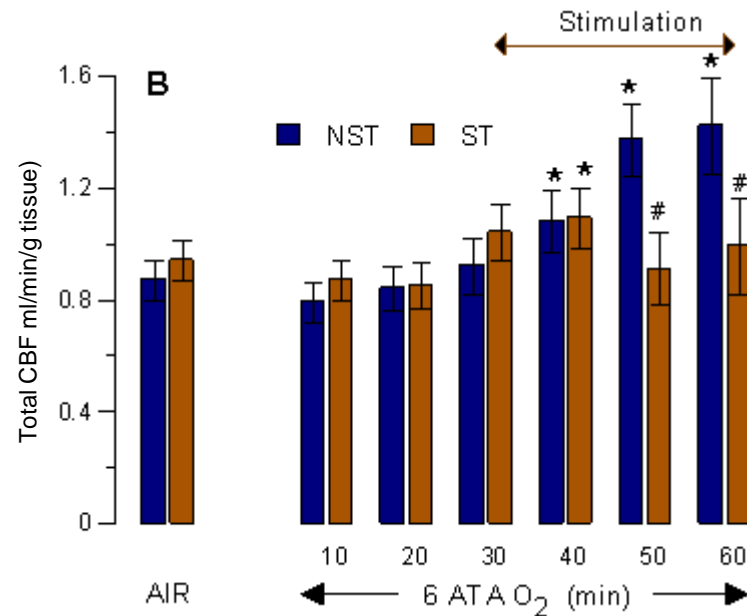
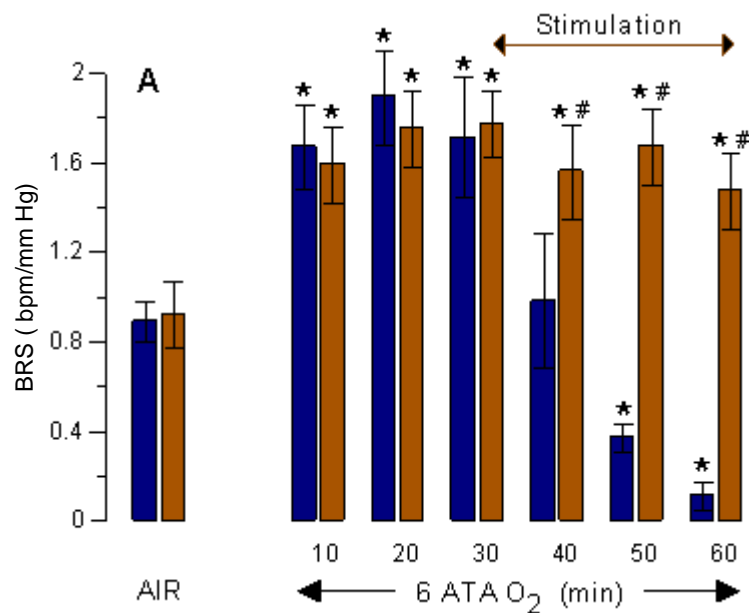
Baroreflex Impairment is Associated with Sympathetic Excitation (A) and Increase in Plasma Norepinephrine (B)



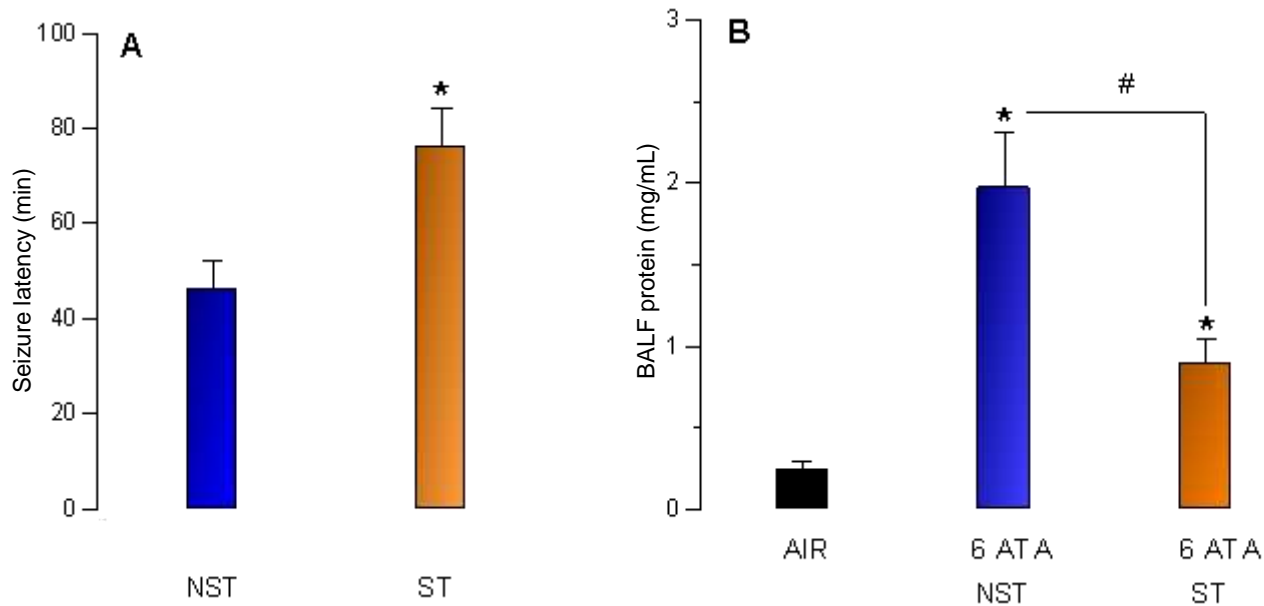
Electrical Stimulation of Aortic Depressor Nerve Prevents Hypertension (A), Tachycardia (B), Sympathetic Excitation (C) and Seizures



Electrical Stimulation of Aortic Depressor Nerve Preserves Baroreflex Function (A) and Prevents Cerebral Hyperemia (B)



Electrical Stimulation of Aortic Depressor Nerve Increases Seizure Latency (A) and Mitigates Lung Injury (B)



Conclusions

Physiological activation of the arterial baroreflex during HBO₂ serves two protective functions:

- Limits convective delivery of oxygen at toxic doses to the brain
- Restrains brain excitability, preventing the development of CNS and pulmonary HBO₂ toxicity
- At < 3 ATA HBO₂-triggered baroreflex sustains depression of sympathetic activity during the entire period of hyperoxia. This baroreflex-derived autonomic response may benefit HBO₂ treatment of patients with sympathetic hyperactivity (e.g. traumatic brain injury, heart failure, hypertension, epilepsy, diabetes).