Noninvasive Optical Detection of Metabolic Distress and Hyperosmolar Treatment Efficacy after Traumatic Brain Injury in Swine

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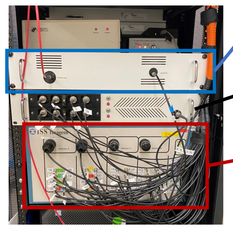
Introduction

- **Traumatic brain injury (TBI) remains a major threat** to Service Member health, operational readiness, and long-term function.
- Following the primary injury, **evolving secondary injury mechanisms can worsen neurological outcomes** (e.g., intracranial hypertension, cerebral edema, hemorrhage, impaired perfusion, and metabolic dysfunction).
- During **prolonged field care**, medics must rely on **indirect or surrogate measures of cerebral status**, limiting real-time clinical decision making
- **Noninvasive optical neuromonitoring** can assess cerebral hemodynamics, oxygen metabolism, and tissue water, with the potential to **identify secondary injury mechanisms and inform care decisions.**

STUDY OBJECTIVES: Characterize cerebral physiological changes after TBI and determine whether optical neuromonitoring can identify clinically relevant secondary injury mechanisms

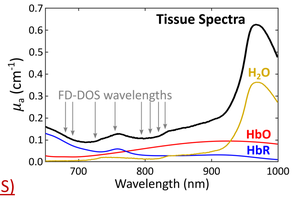
Neurometabolic Optical Monitor (NOM)



Broadband Diffuse Optical Spectroscopy (bDOS)

Diffuse Correlation Spectroscopy (DCS)

Frequency-Domain Diffuse Optical Spectroscopy (FD-DOS)

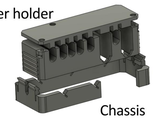


Bottom view

Detector/Source
- bDOS (SDS: 3 cm)
- FD-DOS (SDS: 1.5, 2, 2.5 and 3 cm)
- DCS (SDS: 2.5 cm)

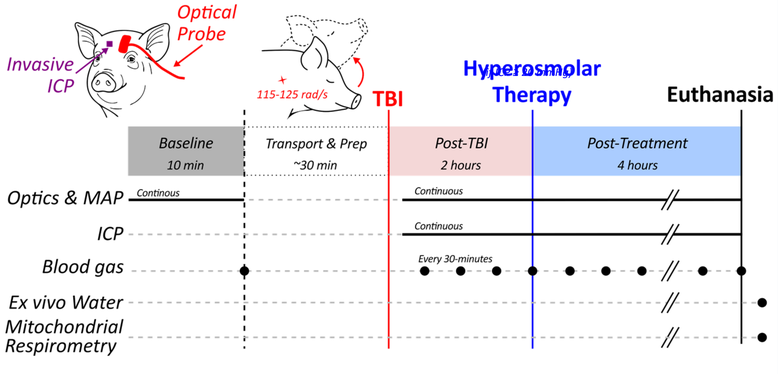
SDS: Source detector separation
HbO: Oxyhemoglobin
HbR: Deoxyhemoglobin

Fiber holder

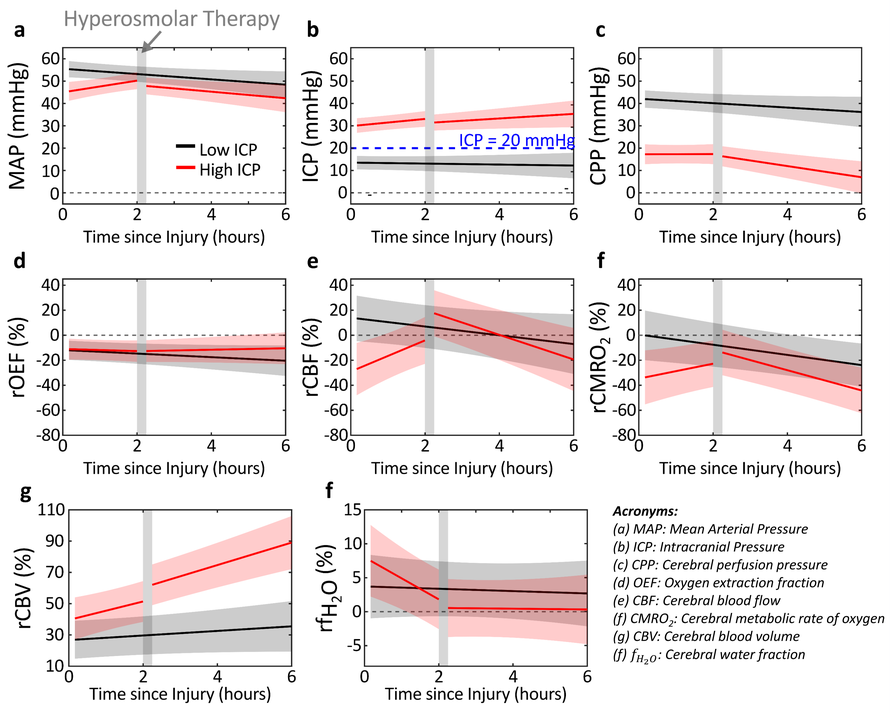


Chassis

Experimental Protocol



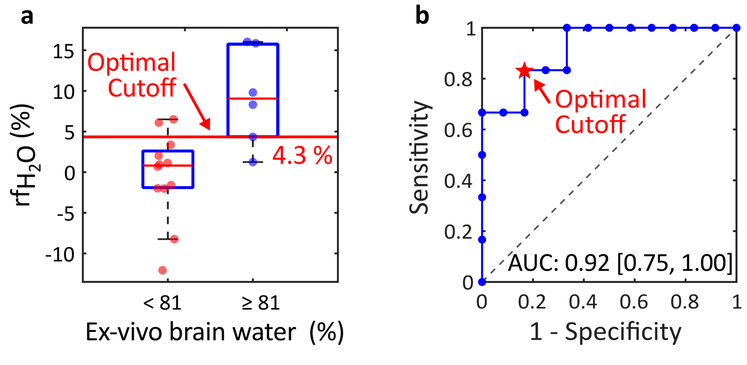
Cerebral physiology and treatment response



Acronyms:
(a) MAP: Mean Arterial Pressure
(b) ICP: Intracranial Pressure
(c) CPP: Cerebral perfusion pressure
(d) OEF: Oxygen extraction fraction
(e) CBF: Cerebral blood flow
(f) CMRO$_2$: Cerebral metabolic rate of oxygen
(g) CBV: Cerebral blood volume
(f) f_{H_2O}: Cerebral water fraction

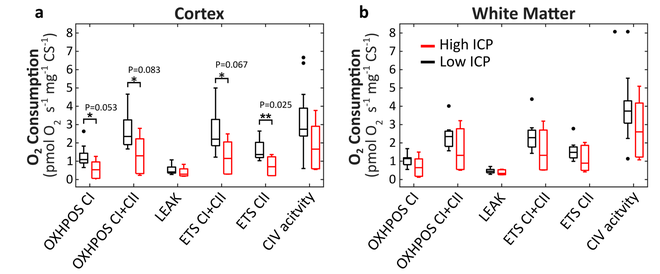
- **~50% of animals developed intracranial hypertension (a) and CPP (c)**
- **Optical monitoring identified reduced CBF/CMRO$_2$ (e and f) and increased CBV (g)**
- **Hyperosmolar therapy lowered ICP transiently (a), with no other clear changes (a-f)**

Optics identified *ex vivo*-confirmed cerebral edema



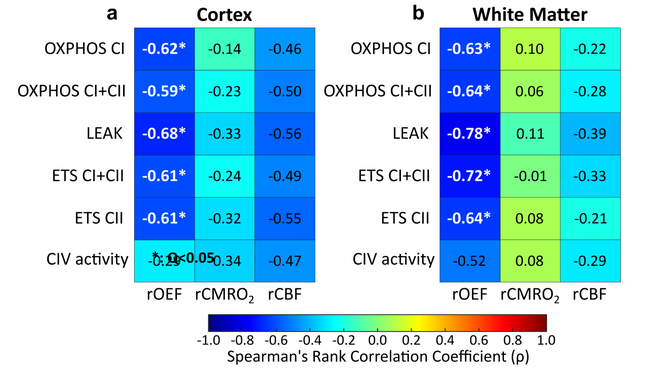
- **Brain water content was greater in animals with *ex vivo*-confirmed edema.**
- **Optical monitoring can separate edema-related changes from blood-volume effects.**

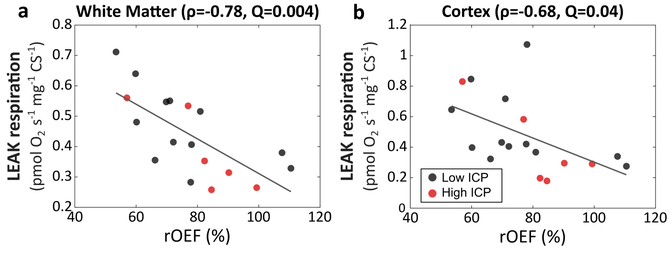
Mitochondrial respirometry at 6 h post-TBI



- **High-ICP animals showed more impaired cortical respiration**
- **Group differences were most evident in cortical tissues**

Optical assessment of mitochondrial injury





- **rOEF showed the strongest associations with LEAK respiration.**
- **Diffuse optics may provide a noninvasive marker of mitochondrial injury.**

Conclusions & Next Steps

- Optical biomarkers reflect hemodynamic and mitochondrial dysfunction
- Diffuse optics has the potential to noninvasively detect cerebral edema
- Noninvasive optical neuromonitoring may support treatment and evacuation decisions during prolonged field care.

NEXT STEPS: Validate biomarkers in field-relevant TBI/polytrauma models and develop brain-focused treatment guidance algorithms.

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