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| 14. ABSTRACT<br><br>Our preliminary data of Year 1 sheds light on the dynamic changes that occur with oxygenation, blood flow, and metabolic responses in the penumbra of the traumatic spinal cord injury site. Spinal cord contusion followed by compression resulted in a prompt loss of perfusion at the sites nearest to the epicentre with a critical reduction of substrate (glucose/oxygen) delivery. Following decompression only partially recovery was observed for blood flow, oxygen and glucose and persisted for several hours. Distal to the injury we also observed a decrease in spinal cord oxygenation although more progressive over time, causing a continual increase in L/P ratio above baseline, suggesting that our experimental SCI caused widespread and sustained hypoxia. Although blood flow levels gradually recovered to baseline levels, however, failed to improve spinal cord oxygenation in our setting. In fact, tissue oxygenation was found to be entirely unaffected, reflected by an elevation in the L/P ratio. This data advances our current understanding of the pathophysiology following spinal cord injury, and in Year 2 we will focus of this study is to evaluate the consequences of hemodynamic support on intraparenchymal blood flow, oxygenation and downstream metabolic responses, after SCI. |              |                          |                            |                                               |                                           |
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## 1 INTRODUCTION

There are currently very few treatments to improve the neurologic outcome of individuals who sustain an acute spinal cord injury (SCI). Treatment options include urgent surgical decompression to relieve pressure on the spinal cord and aggressive augmentation of systemic blood pressure to minimize ischemia. However, improved outcomes from these approaches have not been convincingly demonstrated in randomized or controlled clinical trials, and hence they are not considered 'standards of care'. We postulate that the difficulty in unequivocally demonstrating the benefits of aggressive hemodynamic support may be due to this approach eliciting not only beneficial but also adverse effects on the injured spinal cord, depending upon the presence or absence of spinal cord compression. Our overall objective is therefore to determine how hemodynamic support of mean arterial pressure (MAP) in the presence or absence of spinal cord compression affects the vascular, metabolic, biochemical, and behavioral outcomes of traumatic SCI. We hypothesize that well-intended increases in MAP after decompression contribute to detrimental edema/swelling, hemorrhage, and increased intraparenchymal pressure, in addition to exacerbating ischemia-reperfusion injury mechanisms. To test this hypothesis, we will utilize a novel pig model of SCI, for which we have developed innovative techniques for measuring intraparenchymal spinal cord blood flow (SCBF), hydrostatic pressure, and metabolic responses over time. Achieving a better understanding of how the spinal cord responds to alterations in MAP before and after decompression will provide insights that could be deployed rapidly into clinical practice to optimize the hemodynamic management of acute SCI. Such insights have added significance for soldiers injured in combat where sophisticated therapies are likely not available and the early treatment of their SCI may be limited to basic hemodynamic resuscitation and the management of their MAP.

## 2 KEYWORDS

- Hemodynamic Support
- Spinal Cord Injury Based
- Cord Compression
- Cord Decompression
- Porcine model of SCI
- Spinal Cord Blood Flow
- Microdialysis
- Intraparenchymal Pressure
- Vasopressors

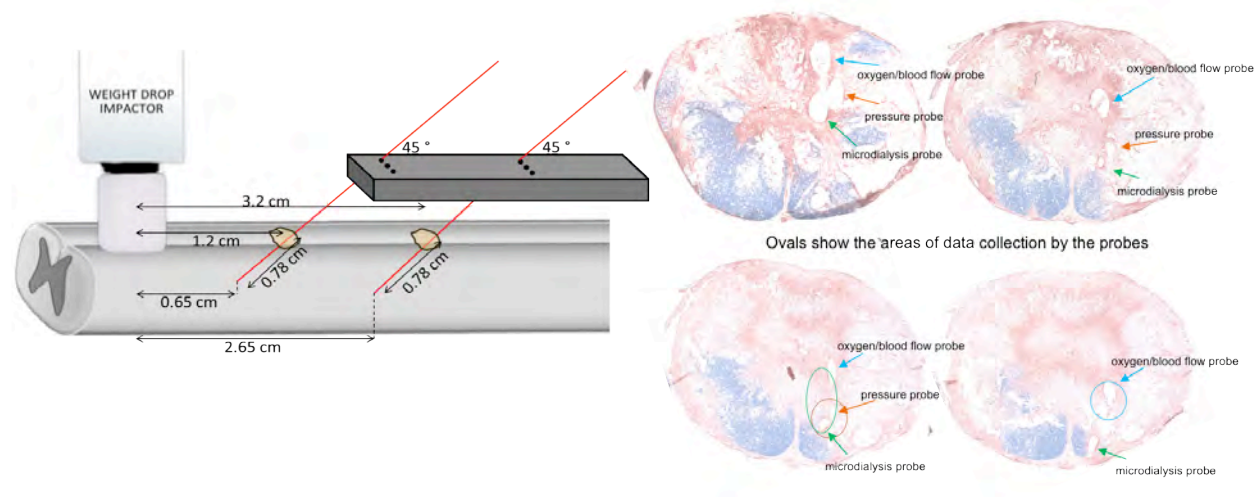
### 3 OVERALL PROJECT SUMMARY

#### 3.1 Methods

We used the porcine model of SCI as developed in our lab involving a combination of contusion and compression components. Using intraparenchymal microdialysis, extracellular fluid from the spinal cord at 1.2- and 3.2-cm caudal to the center of the impact was interrogated for up to 7-days ([Figure 1](#)). These samples were analyzed for various markers of cellular damage, ischemia and energy status, including lactate, pyruvate, L/P ratio and glucose. Continuous monitoring of intraparenchymal spinal cord O<sub>2</sub> tensions (tPO<sub>2</sub>) and blood flow was performed using the Oxylite system from Oxford Optronics. This probe consisted of a 4-channel composite containing a Laser Doppler Flowmetry probe with separate emitting and receiving channels, a fluorescent PO<sub>2</sub> probe, and a thermocouple. As the volume of tissue sampled by our oxygen sensors *in vivo* is estimated to be in the region of 0.5-1mm<sup>3</sup>, the small capillaries within the spinal cord, are likely to contribute more substantially to the SCBF measurements made with laser Doppler flowmetry, then the large vessels on the surface of the cord, such as the anterior/posterior spinal artery.

tPO<sub>2</sub> is defined as the partial pressure of oxygen in tissue and reflects the availability of oxygen for oxidative energy production. tPO<sub>2</sub> represents the balance between oxygen delivery and oxygen consumption. The oxygen portion of the Oxylite probe emits short pulses of blue LED light resulting in a fluorescent discharge that is quenched by tissue O<sub>2</sub>. The signal is received by the Oxylite system and the O<sub>2</sub> tension using a factory-precalibrated algorithm expresses tPO<sub>2</sub> in mmHg. A thermocouple is included in the probe to correct for temperature.

**Figure 1: Triple monitoring fixation device for inserting various sensors into the desired location within the spinal cord.** (A) Illustrates the complete assembled fixation device and how the guides are grouped together into the body portion of the fixation device. (B) In this study, sensors were inserted through the dura 12-mm and 32-mm from the centre of the impact and advanced another 7.8mm at a 30-degree angle. As the impactor is 9.0mm in diameter the final location of the tip of the sensors were located 2.0mm and 22.0mm away from the edge of the impactor. Note: Throughout the report we refer to the sensor location as 12mm and 32mm.



Laser Doppler flowmetry, for measuring relative changes in perfusion, is based on the principle of the Doppler shift: Blood cells traversing tissue are struck by the light and reflect it, whereby the light undergoes a Doppler shift. The surrounding tissue also reflects the light, but in an unshifted manner. Thus the volume of illumination is a mixture of an unshifted and a Doppler shifted component, the magnitude and frequency (wave length) of the latter being related to the number of moving cells and their velocity. Microvascular blood perfusion therefore, is the product of mean blood cell velocity and mean blood cell number concentration present in the small measuring volume of tissue under illumination from the probe.

The technology of the intraparenchymal pressure sensor (FISO company) is based on the Fabry–Perot optical resonator at the end of the fibre, where one of the resonator walls is built as a membrane deflecting under the pressure. Pressure changes inflict different wavelengths being amplified in the reflected spectrum. The conditioner that introduces light into the measuring fibre sensor is additionally equipped with a Charge-Coupled Device matrix for reflected light measurements, and because the relation between membrane deflection and pressure is linear, the pressure is measured by determining the membrane deflection.

Pre-injury measurements were taken 60 minutes before SCI at two distinct caudal regions in the spinal cord (1.2-cm and 3.2-cm from epicentre) as a baseline comparison for all post-injury measurements. These regions were selected to investigate the biochemical and hemodynamic consequences, since others have shown that the initial mechanical impact might have significant effects for a considerable rostro-caudal distance. All pre-injury SCBF and microdialysis values were averaged and normalized to 100%. Post-injury values were expressed as a percentage of their respective baseline values.

## 3.2 Results

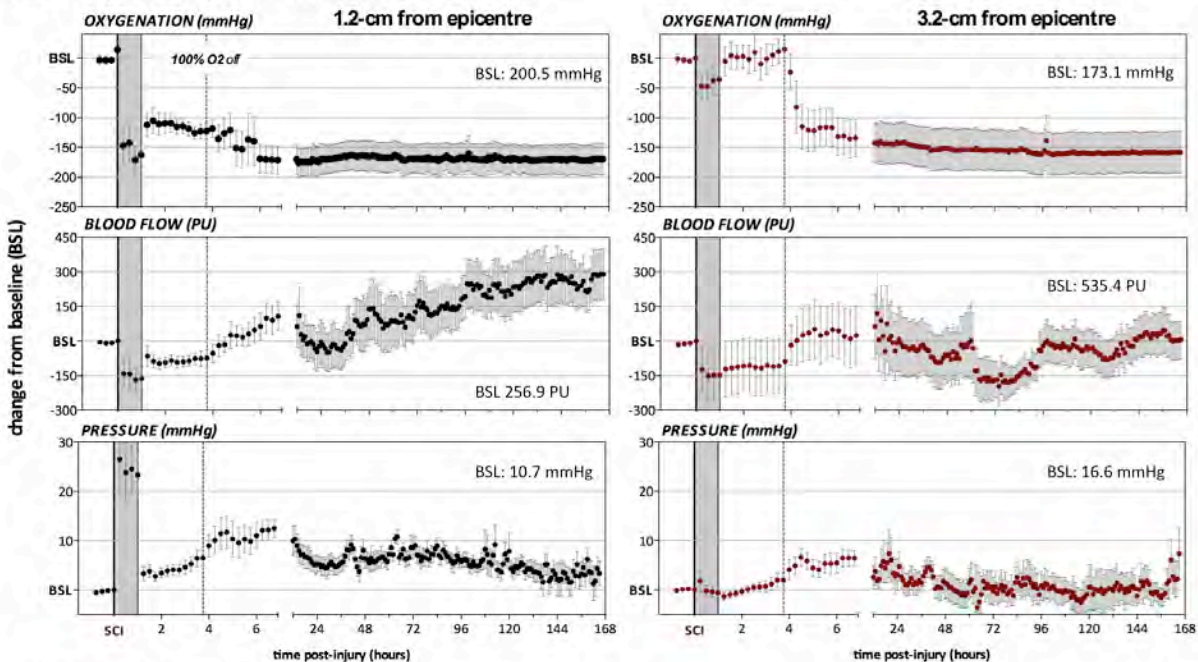
**1.2 cm caudal to the injury epicenter:** Proximal to the injury site (1.2-cm),  $tPO_2$  levels and SCBF fell rapidly below baseline levels following SCI and while the spinal cord remained compressed ([Figure 2A-B](#)). SCBF and  $tPO_2$  levels recovered slightly in the first few minutes after decompression, but remained below average baseline values even after 3 hours of decompression (~25% of baseline for  $tPO_2$  and ~60% of baseline for SCBF). At around 4-6 hours after the SCI, SCBF showed a slight, continuous increase with time to about 150% of the baseline value, while at the same time a dramatic drop in  $tPO_2$  levels was observed to ~8% of baseline. Notably during this time, the animals were weaned off of isoflurane anesthesia, awakened and extubated (Mechanical ventilation was provided with 100% oxygen). Over the time course of the next 2 days, SCBF returned to baseline values. Thereafter, however, a steady increase in SCBF was observed such that blood flow measurements at 7 days were reached 200% of baseline values.  $tPO_2$  levels were fairly constant over the course of the 7-days and remained at 8-10% of baseline values until the end of the study.

During spinal cord contusion and 1-hr of compression, intraparenchymal spinal cord pressure recorded in the 1.2-cm region drastically increased to ~350% ( $\Delta 22$ mmHg) of baseline values and remained elevated throughout the compression period ([Figure 2C](#)). Decompression pressure immediately dropped intraparenchymal pressure back to within baseline values. After

this transient fall, pressure gradually increased once again compared to baseline reaching a maximum of ~220% ( $\Delta 14\text{mmHg}$ ) 12 at 4.5-hrs post-SCI. At 15-hrs a slight decrease in pressure was observed although values remained above baseline levels (150%;  $\Delta 5\text{mmHg}$ ). Throughout the remained of the study, pressure always remained well above baseline (150%).

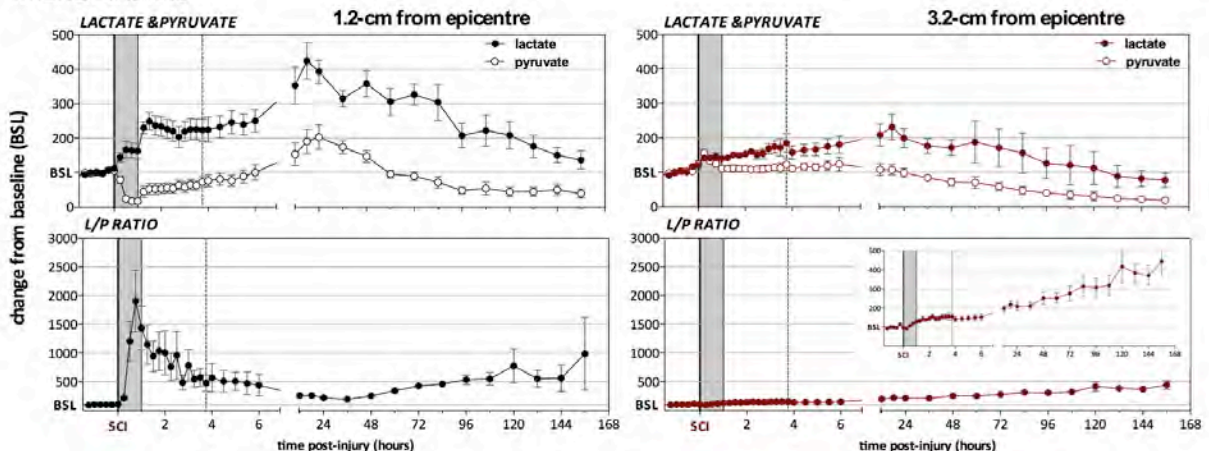
**Figure 2: Disturbances in the oxygenation and spinal cord blood flow (SCBF) monitored with intraparenchymal inserted Oxylite sensors for 7 days after SCI.** intraparenchymal (A) intraparenchymal oxygenation response, (A') similar oxygenation data as depicted in (A), however with adjusted y-scale, (B) intraparenchymal blood flow response, (C) intraparenchymal pressure response.

#### OXYLITE, OXYFLO, FISO SENSORS



**Figure 3. Disturbances in the metabolism of energy-related substrate monitored with microdialysis technique for 7 days during spinal cord contusion followed by compression and decompression in a pig model of SCI.**

#### MICRODIALYSIS





With respect to the spinal cord metabolism as measured by microdialysis ([Figure 3](#)), glucose values decreased significantly upon SCI, and subsequently returned to baseline by day 1. Lactate to pyruvate (L/P) ratio, a marker for tissue ischemia, increased significantly within minutes after SCI and cord compression and subsequently decreased after decompression. However, the L/P ratio increased again to levels 5-fold above baseline by day 7.

**3.2 cm caudal to the injury epicenter:** Distal from the injury site (3.2-cm), the response to SCI, compression and decompression was minimal, however, over the course of 7-days, gradual changes were observed. Glucose levels began to fall below baseline around 5 days post-injury. In addition, oxygenation and SCBF decreased slowly but continuously over time, while the L/P ratio steadily increased up to 4-fold above baseline. Furthermore, it could be observed that there was a slightly increase in intraparenchymal pressure at 1.5-hrs.

### 3.3 Conclusion and discussion

**Early metabolic and hemodynamic responses to SCI (within hours):** Taken together, our preliminary data sheds light on the dynamic changes that occur with oxygenation, SCBF, and metabolic responses in the penumbra of the traumatic spinal cord injury site. Spinal cord contusion followed by compression resulted in a prompt loss of perfusion at the sites nearest to the epicentre with a critical reduction of substrate (glucose/oxygen) delivery. Following decompression only partially recovery was observed for blood flow, oxygen and glucose and persisted for several hours, which may be attributable to a variety of vascular events, including vessel rupture, occlusive thromboses, loss of vascular autoregulation and/or vasospasm. The immediate increase in spinal cord lactate concentration and L/P ratio following injury may be secondary to tissue hypoperfusion, hypoxia, and anaerobic glycolysis caused by the primary injury, reflecting a redox state where the lactate dehydrogenase reaction is shifted towards lactate as a result of an inadequate supply of oxygen and glucose. However, increased lactate synthesis may also partially be explained as a result of excessive glutamate-mediated neuronal activation, as well as glycolysis activity of shed erythrocytes, which can produce quite large amounts of lactic acid. The lactate diffuses out of the cells and is converted to pyruvate and then aerobically metabolized to carbon dioxide and ATP. Under particular circumstances the brain has the capacity to use blood-derived lactate in this manner.

Although lactate extrusion into the extracellular space was classically believed to be a metabolically waste product of anaerobic metabolism, there is increasing evidence that it is a key intermediary in normal metabolic pathways. Lactate fuels energy-requiring processes and, in this context, astrocytes are a major source of lactate production in response to glutamate released by neurons. This lactate may become a favored fuel for neurons, in particular after traumatic or hypoxic injury to the CNS, even during conditions of preserved aerobic glycolysis. While variations in opinion still exist, the general viewed as a negative metabolic event, has shifted to an essential compound of neuronal functioning.

**Late metabolic and hemodynamic responses to SCI (within days):** Discontinuation of anesthesia and ventilation at the end of the surgical procedure coincided with increased spinal cord perfusion proximal to the injury and fully recovered to baseline levels relatively quickly after tracheal extubation, with an subsequent rise by day 2 resulting in a 2-fold increased compared to baseline at day 7. Although glucose levels gradually recovered to baseline levels during the time of increased perfusion, however, failed to improve spinal cord oxygenation in our setting. In fact, tissue oxygenation was found to be entirely unaffected. Although, the increased blood flow may superficially increase the conventional definition of delivery of oxygen, it does not improve mean tissue oxygenation, reflected by an elevation in the L/P ratio (notably: values are less high compared to immediately after SCI).



Blood flow is closely coupled to tissue metabolic activity in most organs and tissue of the body. Increases or decreases in metabolism lead to increases or decreases in the release of vasodilator substances. This ensures that the tissue is adequately supplied and that products of metabolism (e.g. CO<sub>2</sub>, H<sup>+</sup>, lactate) are removed. Another mechanism that may couple blood flow and metabolism involves changes in the partial pressure of oxygen (tPO<sub>2</sub>). Decreased tissue tPO<sub>2</sub> resulting from reduced oxygen supply or increased oxygen demand causes vasodilation. Hypoxia-induced vasodilation may be direct (inadequate O<sub>2</sub>) or indirect via vasodilator by-products of metabolism. This metabolic theory of blood flow regulation may explain at least in part our observation of increased perfusion at 2 days post-injury.

Distal to the injury (3.2-cm from epicenter) we also observed a decrease in spinal cord oxygenation although more progressive over time, causing a continual increase in L/P ratio above baseline, suggesting that our experimental SCI caused widespread and sustained hypoxia. However this decreased oxygenation and increased hypoxia did not coincide with a sharp increase in spinal cord perfusion, actually SCBF seemed rather stable throughout the experiment. Notably the level of oxygenation did not reach similar low levels as observed proximal to the injury site until at least day 4. This might suggest that the relationship between oxygenation and blood flow is independent of cell oxygenation until a critically low cell tPO<sub>2</sub> is achieved.

## 4 KEY RESEARCH ACCOMPLISHMENTS

- Distal to the impact, a clearly increased cord pressure is observed within hours after SCI
- A delayed rostro-caudal increase in cord pressure was observed
- Ischemia and hypoxia are observed within minutes after SCI
- Recovery of cord perfusion is observed within 12 hours after SCI.
- Hypoxia remains evident throughout the duration of the experiment (7-days)
- Hypoxia slowly progresses proximally

## 5 CONCLUSIONS

This research focuses on the acute hemodynamic management of spinal cord injury and specifically addresses the current practice of aggressively maintaining the mean arterial pressure (MAP) at 85-90 mm Hg for 5-7 days post-injury. This has particular relevance to military personnel who are injured in combat zones. Our data advanced our current understanding of the pathophysiology following spinal cord injury, and in Year 2 we will focus of this study is to evaluate the consequences of hemodynamic support on intraparenchymal SCBF, oxygenation and downstream metabolic responses, after SCI.

## **6 PUBLICATIONS, ABSTRACTS, PRESENTATIONS**

Poster presentation, Society for Neuroscience 2015, Chicago, Illinois, Oct 17-21:

Elena Okon, Femke Streijger, Neda Manouchehri, Kitty So, Katelyn Shortt, Brian K. Kwon (2015) *Changes in Spinal Cord Energy Metabolism, Oxygenation and Blood flow After SCI Using a Porcine SCI Model.*

## **7 INVENTIONS, PATENTS AND LICENSES**

Nothing to report

## **8 REPORTABLE OUTCOMES**

Nothing to report

## **9 OTHER ACHIEVEMENTS**

Nothing to report

## **10 REFERENCES**

None

## **11 APPENDICES**

None