Scientific Research Publishing

Open Journal of Veterinary Medicine, 2025, 15(12), 308-318
https://www.scirp.org/journal/ojvm
ISSN Online: 2165-3364
ISSN Print: 2165-3356

Evaluating Catecholamines and Blood Pressure in Traumatic Spinal Cord Injury Utilizing Non-Human Primate Model

—Pathological and MRI Correlation in TSCI

Heather A. Simmons[1], Kevin M. Johnson[2], Timothy Hacker[3], Dane Schalk[1], Kevin Brunner[1], Puja Basu[1], Casey Fitz[1], Saverio Capuano III[1], Jennifer M. Hayes[1], Shanker Nesathurai[4*]

[1]Wisconsin National Primate Center, University of Wisconsin, Madison, WI, USA
[2]Department of Medical Physics, School of Medicine and Public Health, University of Wisconsin, Madison, WI, USA
[3]Department of Medicine, School of Medicine and Public Health, University of Wisconsin, Madison, WI, USA
[4]Michael G. DeGroote School of Medicine, McMaster University, Hamilton, Canada
Email: *nesathurai@hhsc.ca

How to cite this paper: Simmons, H.A., Johnson, K.M., Hacker, T., Schalk, D., Brunner, K., Basu, P., Fitz, C., Capuano III, S., Hayes, J.M. and Nesathurai, S. (2025) Evaluating Catecholamines and Blood Pressure in Traumatic Spinal Cord Injury Utilizing Non-Human Primate Model. *Open Journal of Veterinary Medicine*, **15**, 308-318.
https://doi.org/10.4236/ojvm.2025.1512020

Received: November 5, 2025
Accepted: December 8, 2025
Published: December 11, 2025

Copyright © 2025 by author(s) and Scientific Research Publishing Inc.
This work is licensed under the Creative Commons Attribution International License (CC BY 4.0).
http://creativecommons.org/licenses/by/4.0/
Open Access

Abstract

There are significant gaps in the understanding related to the biochemical, pathophysiological and radiological features of traumatic spinal cord injury in the first hour post-injury. In this context, a validated experimental traumatic spinal cord injury non-human primate model was utilized to evaluate serum catecholamine levels and blood pressure response during this hyperacute time frame. The subject was a single cynomolgus macaque monkey. The lesion was created with an epidural balloon catheter. The serum dopamine, epinephrine, and norepinephrine levels increased subsequent to the deflation of the catheter. The blood pressure, as measured from the aorta at the level of injury, decreased immediately after injury and remained below the baseline level. The histological abnormalities included scattered angular neurons with mild to moderate multifocal vacuolization of the neuropil. This non-human primate model can be utilized to further understand the pathophysiology of traumatic spinal cord injury.

Keywords

Spinal Cord Injury, Catecholamines, Blood Pressure, Pathology, MRI, Radiology

1. Introduction

Worldwide, there are approximately 15 million people living with spinal cord in-

jury [1]. Traumatic spinal cord injury (TSCI) has a profound short-term and long-term impact on the person injured as well as their families and friends.

There are significant gaps in data concerning the biochemical, pathophysiological and radiological features of TSCI in human beings during the first hour post-injury ("hyperacute period"). This is related, in part, to the time that it takes to medically stabilize and safely transport a patient from the scene of an accident to the hospital. As such, investigations, including biochemical laboratory and advanced radiological studies of TSCI, are typically completed later in the course of clinical presentation. Due to these operational realities, the biochemical and physiological response to injury is not well delineated. In this context, animal models of TSCI can provide meaningful insights into advancing scientific knowledge during this very early time frame.

This paper reports on how a validated NHP model of TSCI can be utilized to further the understanding of TSCI. The experimental spinal cord injury is created with an epidural balloon catheter. This method has mechanistic and pathological features that are similar to human TSCI. From a mechanistic perspective, the balloon was rapidly inflated, which is analogous to the rapid transfer of energy in human TSCI. The balloon remained inflated for fixed period of time; this is analogous to the residual displacement of spinal cord, such as disk material, bony fragments, or hematoma. From a pathological perspective, the experimental lesions created by this method do not compromise the dura mater, which is consistent with most human injuries [2]-[5]. In this experiment, serum catecholamine levels were assayed before and after balloon catheter injury. Additionally, blood pressure was measured from the aorta before and after the experimental TSCI; measurement from this location may serve as surrogate of perfusion at the level of injury. Finally, the histological features at the level of experimental TSCI are described.

2. Materials and Methods

The subject was an adult female cynomolgus macaque (*Macaca fascicularis*). The animal was 19.7 years old and weighed 5.36 kilograms when assigned. The experimental protocol was approved by the University of Wisconsin at Madison Institutional Animal Use and Care Committee. Experimental procedures were completed under general anesthesia with appropriate analgesia and sterile surgical technique. The animal was sedated with a combination of ketamine (10 mg/kg) and midazolam (0.2 mg/kg) administered intramuscularly. Intravenous access was established by placing a 20-gauge catheter in the left cephalic vein. Anesthetic induction was facilitated by use of intravenous propofol bolus (1 - 2 mg/kg). Topical lidocaine spray was administered to the vocal folds prior to placement of a 4.0 mm endotracheal tube. Anesthesia and analgesia were maintained with isoflurane inhalant (0.75%) with continuous rate infusions of fentanyl (1 - 2 mcg/kg/hr) and ketamine (5 - 10 mcg/kg/min). Fentanyl bolus (0.5 mcg/kg) was administered four times during the procedure as indicated for analgesia. Isotonic fluids (1 - 3 mL/kg/hr) were administered throughout the procedure and a single bolus (5 mL/kg) was used

to address an episode of hypotension. A dose of heparin sodium (100 units) was administered intravenously prior to the procedure. A dose of hydromorphone (0.1 mg/kg) was administered intravenously following completion of the procedure and discontinuation of fentanyl. Gadobenate dimeglumine (150 mg/kg) was administered intravenously prior to MR imaging.

A small laminotomy was performed at the level of the fifth lumbar vertebra. An epidural balloon catheter was inserted and advanced approximately 10 cm cranial, to the level of the lower thoracic spinal cord.

A "cut down" was completed to identify the right femoral artery and vein. A digital blood pressure transducer was inserted into the femoral artery and advanced to the aorta. With fluoroscopic guidance, the transducer was placed at the same level as the epidural balloon. A catheter was inserted into the femoral vein to obtain blood samples.

The balloon was inflated rapidly; this corresponds to the initial traumatic forces applied to the spinal cord. The balloon remained inflated for 45 minutes. The initial rapid inflation corresponds to the initial mechanical injury with transmission of forces to the spinal cord and related structures. The continued inflation of the balloon corresponds to residual displacement of the spinal cord secondary to hemorrhage, bony fragments, and disk material. Additional details regarding the method of experimental lesion creation are noted elsewhere [4] [5].

The balloon was then deflated. The catheter was removed, and the surgical incision was closed.

MRI imaging was completed with a GE 3 Tesla Discovery machine. Sagittal and axial images of the spinal cord were obtained. The images were obtained with and without intravenous contrast.

Serum catecholamines (*i.e.*, dopamine, norepinephrine, and epinephrine) levels were obtained immediately prior to inflation of the balloon and immediately after inflation. Serum catecholamines were also measured immediately after balloon deflation. Catecholamine levels were also measured prior to euthanasia and necropsy; this blood draw included an additional test for normetaephrine and metanephrine.

The subject was then humanely euthanized with the administration of sodium pentobarbital (at least 50 mg/kg intravenously). A postmortem examination was immediately conducted. The time from balloon inflation to necropsy was approximately 4 hours and 45 minutes. For histopathology, spinal cord sections were collected at the epicenter of the lesion with systematic sectioning of the entire cord at approximately 1 cm intervals both caudal and cephalad. Tissue sections were fixed in 10% neutral buffered formalin for a minimum of 7 days, routinely processed and embedded in paraffin. 5 μm slices were stained with hematoxylin and eosin. The tissues were stained with Luxol fast blue with Periodic Acid-Schiff counterstain (LFB/PAS) for a comparison of histologic changes. A subset of spinal cord slides from this animal was collected, fixed, and trimmed. These slides were compared to previously completed experiments that included: an uninjured control

monkey, a monkey where the balloon was inflated for a period of 1 min, and a monkey where the balloon was inflated for a period of 60 min.

3. Results

The blood pressure values were obtained by averaging the traducer readings over a one-minute period (Table 1). Three minutes post-deflation, in the context of the low pressure, a bolus of approximately 25 mL of normal saline was administered.

Table 1. Blood pressure measured from the aorta.

Time	Arterial BP	Mean Arterial Pressure	Heart Rate
Prior to creation of lesion	106/53	70	130
1 min post-balloon inflation	109/60	76	152
3 min post-balloon inflation	99/56	70	149
5 min post-balloon inflation	108/63	78	149
20 min post-balloon inflation	94/46	62	154
30 min post-balloon inflation	90/43	59	156
45 min post balloon inflation	80/35	50	158
1 min post-balloon deflation	68/30	43	149
3 min post-balloon deflation	52/23	33	135
5 min post-balloon deflation	64/25	38	134
20 min post-balloon deflation	81/36	51	150
30 min post-balloon deflation	85/39	55	153

Serum catecholamine levels were obtained before and after inflation of the balloon catheter (Table 2). Serum normetaphrine and metaphrine levels were also obtained immediately prior to euthanasia. The values were 1.52 and 94 nmol/L respectively.

There are no established normal values for dopamine, norepinephrine, epinephrine, normetaphrine, or metaephrine in non-human primates.

Table 2. Serum catecholamine levels in nmol/L.

Time	Dopamine	Norepinephrine	Epinephrine
Immediately prior to inflation	<20	174	672
Immediately post-inflation	33	195	533
Immediately post-deflation	125	204	1086
Prior to necropsy	706	1763	1579

Histologic Evaluation

In this experiment, the balloon was inflated for a period of 45 minutes.

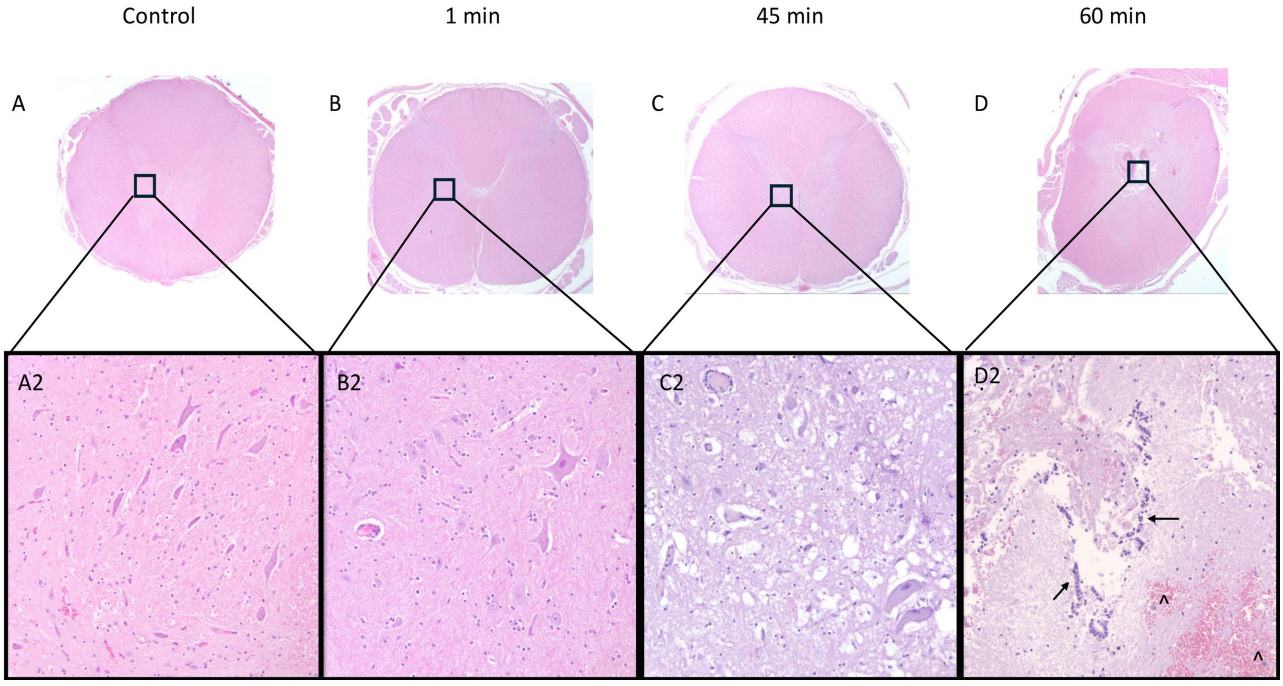
Hematoxylin and Eosin Stain

At the level of injury, cross-sections of the spinal cord had no detectable changes (control and 1 min balloon inflation), scattered angular neurons with mild to moderate multifocal vacuolization of the neuropil (45 min), and severe neuropil compression with disruption of the central canal and cord architecture with multiple large regions of hemorrhage (60 min.). Please see **Figure 1**.

Luxol Fast Blue with Periodic Acid-Schiff Counterstain

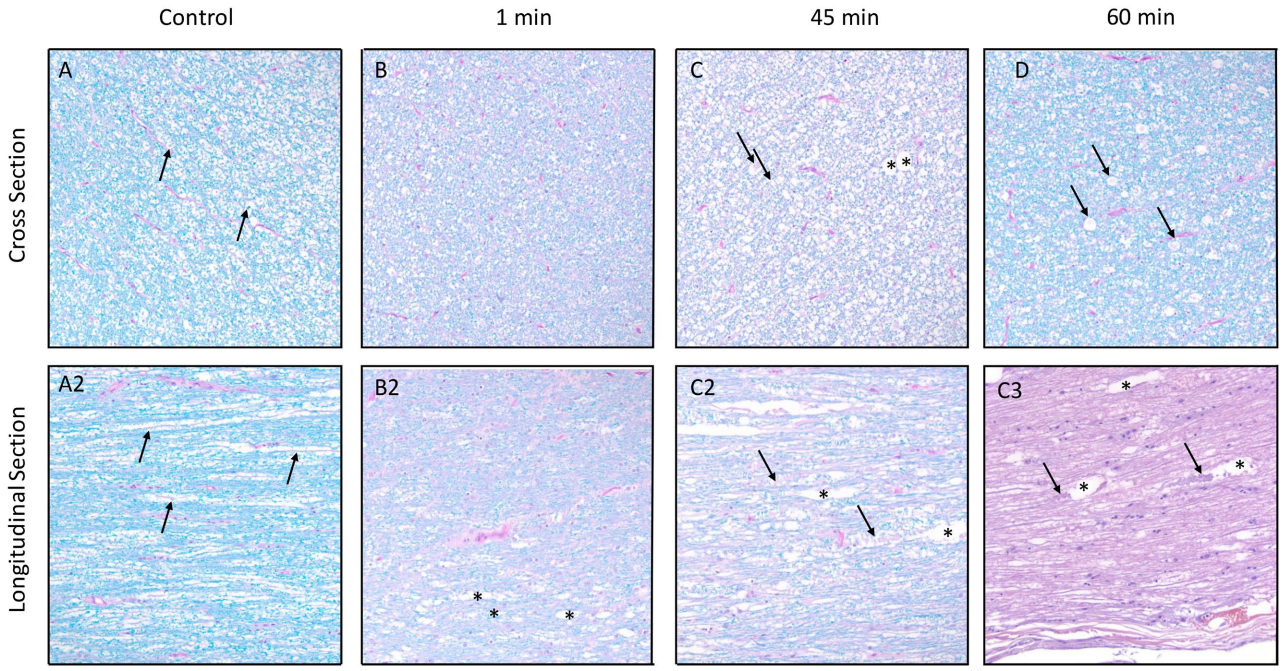
At the level of injury, sections of spinal cord stained with LFB/PAS had no detectable changes (control), minimal scattered mildly dilated myelin sheaths (1 min balloon inflation), multiple moderately dilated myelin sheaths with scattered swollen axons (45 min balloon inflation), and more numerous dilated myelin sheaths with swollen axons (60 min balloon). Note: a longitudinal section of spinal cord at the level of ballon inflation was not available after 60 minutes due to the degree of tissue disruption. Please see **Figure 2**.

There were no discernable abnormalities on MRI imaging within this region of the spinal cord after 45 minutes of balloon compression.



Legend: Photomicrographs of spinal cord stained with hematoxylin and eosin at 2× magnification (A) - (D) and 20× magnification of grey matter regions (A2) - (D2) indicated by black boxes in (A) - (D). Cross-sections of thoracic spinal cord (A), (A2), (B), and (B2) have no significant histologic changes. Section (C2) has moderate multifocal neuropil vacuolization, loss of cellular detail within neurons, and a single perivascular mononuclear cuff in the upper left portion of the photomicrograph. Section (D) has marked lateral compression of both the white and grey matter with disruption of cord architecture. Higher magnification (D2) has a discontinuous line of ependymal cells (arrows) indicating rupture of the central canal, the disruption of neuropil architecture, and multiple foci of hemorrhage (^).

Figure 1. HE stained cross-sections of spinal cord at the site of balloon inflation.



Legend: Photomicrographs of spinal cord stained with LFB/PAS and one HE stained section at 20× magnification. Cross and longitudinal sections of control spinal cord ((A) & (A2)) show consistently sized axons (arrows). The cross-section of the spinal cord after one minute of balloon inflation (B) was unremarkable. The longitudinal section (B2) has rare scattered mildly dilated of myelin (*). Both cross (C) and longitudinal (C2) sections of spinal cord after 45-minute balloon inflation have small numbers of dilated myelin sheaths (*) and occasional swollen axons (arrows). The HE-stained longitudinal section also demonstrates swollen axons (arrows) and dilated myelin sheaths (*). The cross-section of spinal cord at 60 minutes (D) has more numerous swollen axons in dilated myelin sheaths (arrows). A longitudinal section of spinal cord from the 60-minute experiment was not available for comparison.

Figure 2. Cross-sections and longitudinal sections of thoracic spinal cord at the site of balloon inflation.

4. Discussion

At a general caveat, the inferences and generalizations made from an experiment involving one subject require some level of circumspection. In animals, there is variability in physiological responses to perturbations and/or treatments. Additional experiments that include more subjects are needed to allow for robust statistical evaluation. Nevertheless, to our knowledge, this is the only published data on the serum catecholamine levels as well as blood pressure during the hyperacute time period of non-human primate model of TSCI. We also believe that this is the only published data that measures blood pressure from the aorta at the level of experimental traumatic spinal cord injury.

In human beings, there is typically a drop in blood pressure during the early phase of TSCI. The blood pressure is usually measured by a blood pressure cuff on the arm. It is assumed that the blood pressure reading from the limb is a proxy for arterial blood supply to the spinal cord. However, based on a review of the literature, this construct has not been established.

In this single subject, the experimental spinal cord injury resulted in decrease in systolic and diastolic blood pressure, as measured at the aorta. The pressure recording device was positioned at the same anatomical level as the experimental

TSCI.

It is reasonable to infer that blood pressure readings at the aorta may be a proxy of the perfusion pressure within the radicular arteries that supply the spinal cord. However, occlusion and/or spasm to vessels distal to the point of measurement can affect the blood pressure at the microcirculatory level of the injured spinal cord.

During the post-deflation period, the blood pressure dropped to 52/23. At this point, a bolus of 25 cc of normal saline was administered. The blood pressure partially recovered when the balloon in the epidural space was deflated but remained below the pre-lesion level until the time of euthanasia.

It has been postulated that vascular mechanisms contribute to hemorrhagic necrosis of the spinal cord, subsequent to traumatic injury [6]. The decrease in blood pressure at the aorta, which was measured at the level of injury, is consistent with this hypothesis.

The experimental spinal cord lesion also resulted in an increase in serum dopamine and norepinephrine levels at the post-deflation time point, when compared to pre-lesion values. The dopamine, norepinephrine and epinephrine continued to increase up to the time of euthanasia (approximately four and half hours post balloon inflation). The metanephrine and normetaphrine levels were also obtained from the blood obtained at a single time point immediately prior to euthanasia; these are metabolites of epinephrine and norepinephrine, respectively. In the context of the pre- and post-lesion data, it is reasonable to infer that the post-lesion increase in dopamine, norepinephrine and epinephrine is related to the experimental TSCI. Of note, there are no published normative serum dopamine, norepinephrine, epinephrine, metaphrine or normetaphrine levels in NHP. Nevertheless, a component of the increase in catecholamine levels could be the result of the ongoing manipulation of the subject during the experimental procedure.

In this experiment, the balloon was inflated for a period of 45 minutes. The histologic findings at the level of injury demonstrated in this experiment demonstrated mild to moderate vacuolization of the neuropil, moderately dilated myelin sheaths, and scattered swollen axons. In a previous experiment, where the balloon was inflated for one hour, there was disruption of the spinal cord architecture. At this one-hour time point, there were multiple regions of hemorrhage and more numerous dilated myelin sheaths as well as a greater number of swollen axons.

The role of catecholamines in modulating TCSI has been a subject of historical debate. In the 1970s, TSCI studies in cats reported increased norepinephrine in the spinal cord tissue [7]-[9]. Osterholm *et al.* argued that surge in norepinephrine contributed to spinal cord compromise during the hyperacute period. They coined the term "hemorrhagic necrosis" to characterize the histopathological appearance of the injured spinal cord (hemorrhage, cellular pallor, and gross disruption of the cytoarchitecture). These pathological findings were noted as early as 1-hour post-experimental TSCI in cats [7] [8]. In this subject, there was disruption of the histologic architecture of the spinal cord. In a previously published paper,

where the balloon was inflated for one hour, there were even more striking histologic abnormalities [10] [11].

Of note, the finding that norepinephrine was elevated in cats with experimental TSCI is subject to some disagreement [12]. Other laboratories have not been able to confirm an elevation in this norepinephrine in cats [13] [14] or dogs [14]. There have been reports of dopamine levels, a precursor of norepinephrine, being increased in TSCI in cats [15] and dogs [14].

The elevated catecholamine levels may be due to the release by neurons within the central nervous system and/or alternatively from the adrenal medulla. In health, adrenal medulla produces approximately 10 percent of the serum norepinephrine [16]. However, there is evidence from experiments in adrenalectomized cats that the source is primarily the adrenal medulla [9]. In the context of the limited animal data, the predominate source of serum catecholamines in TSCI warrants further research.

We have not been able to identify NHP studies that evaluated serum catecholamines during the hyperacute phase of TSCI. However, in 1975, Bingham *et al.* evaluated the level of norepinephrine levels in TSCI spinal cord tissues of rhesus monkeys after experimental TSCI [17]. The experimental lesions were created in the mid-thoracic spinal cord with Allen's method. Tissue norepinephrine and dopamine levels in the spinal cord were determined spectrophotofluorometrically on pulverized tissues as described by Shellenberger and Gordon. Norepinephrine and Dopamine levels were not increased. Epinephrine levels were not evaluated.

Animal studies have suggested that medications that deplete catecholamines, such as reserpine, may decrease the histopathological abnormalities associated with TSCI. Alpha blockers and dopamine blockers have been reported to decrease histopathological abnormalities [7].

The lack of MRI abnormalities noted in this subject after balloon inflation for 45 minutes is consistent with previous experiments [10] [11]. Specifically, MRI abnormalities were not present when the balloon was inflated for periods of 1 minute, 5 minutes, or 20 minutes in previous experiments [11]. MRI abnormalities have been noted when the balloon remains inflated for 60 minutes [10].

We have not been able to identify any published studies on serum catecholamine levels in human TSCI during the hyperacute period. There is evidence that serum catecholamine levels are altered in chronic spinal cord injury [18] [19]. Patients with spinal cord injury have manifestations of dysautonomia with features of pikothermia, piloerection, priapism and sweating abnormalities. Some patients experience autonomic dysreflexia, which is a medical emergency [20].

Blood pressure in human TSCI may be profoundly decreased in the hyperacute period. The "Best Practice Guidelines", which are promulgated by the American College of Surgeons, recommend intravenous fluids to treat hypotension. The next line of treatment is "…vasopressors as needed". The guidelines state an "alpha- and beta-adrenergic activity is recommended" [21]. In this context of the data presented

here, additional research into this approach may be helpful.

One unanswered paradox relates to the apparent decrease in blood pressure, notwithstanding the elevated levels of serum catecholamines. In human beings with TSCI, the decrease in blood pressure is attributed to "spinal shock". Mechanistically, this is attributed to the traumatic disruption to the autonomic nerve fiber tracts that result in impaired the facilitation of the blood pressure response. Spinal shock is one manifestation of the dysautomonia associated with TSCI [22].

In this subject, there was flocculent debris in the central canal. In another experiment, where the balloon was inflated for 1 hour, debris was also noted in the central canal [11]. Speculatively, this debris may be composed of cellular and/or blood products. We speculate that this debris in the central canal may induce a metabolic response that modulates blood pressure control centers within the brainstem. Alternatively, the experimental spinal cord injury impairs ascending and/or descending sympathetic tracts, which results in impaired blood pressure response.

In cats with experimental spinal cord injury, it has been demonstrated that hemorrhagic necrosis can be minimized by lesioning of spinal cord fibers above the level of injury. It has also been demonstrated that lesioning the dorsal root ganglia at the level of injury, prior to experimental TSCI, can also minimize hemorrhagic necrosis [7]. The lesioning procedures are likely to reduce the amount of catecholamines at the site of injury. This observation supports the construct that descending modulation contributes to the histopathological changes in TSCI.

The data from this preliminary experiment suggest that catecholamine levels are increased, and blood pressure is decreased during the hyperacute period of TSCI. Conceptually, elevated catecholamines would lead to an increase in blood pressure. A clinical paradox is worthy of further investigation. A paradox where animal models may incrementally advance our understanding of human TSCI.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] World Health Organization (2024) Spinal Cord Injury. https://www.who.int/news-room/fact-sheets/detail/spinal-cord-injury

[2] Miller, A.D., Westmoreland, S.V., Evangelous, N.R., Graham, A., Sledge, J. and Nesathurai, S. (2012) Acute Traumatic Spinal Cord Injury Induces Glial Activation in the Cynomolgus Macaque (*Macaca fascicularis*). *Journal of Medical Primatology*, **41**, 202-209. https://doi.org/10.1111/j.1600-0684.2012.00542.x

[3] Nesathurai, S., Graham, W.A., Mansfield, K., Magill, D., Sehgal, P., Westmoreland, S.V., *et al.* (2006) Model of Traumatic Spinal Cord Injury in *Macaca fascicularis*: Similarity of Experimental Lesions Created by Epidural Catheter to Human Spinal Cord Injury. *Journal of Medical Primatology*, **35**, 401-404. https://doi.org/10.1111/j.1600-0684.2006.00162.x

[4] Seth, N., Simmons, H., Masood, F., Graham, W.A., Rosene, D.L., Westmoreland, S., *et al.* (2018) Evidence from a Non-Human Primate Model of Traumatic Spinal Cord Injury in Cynomolgus Macaques (*Macaca fasicularis*) to Evaluate for the Efficacy of

a Combination Pharmacological Treatments. *Comparative Medicine*, **68**, 63-73.

[5] Sledge, J., Andrew Graham, W., Westmoreland, S., Sejdic, E., Miller, A., Hoggatt, A., *et al.* (2013) Spinal Cord Injury Models in Non-Human Primates: Are Lesions Created by Sharp Instruments Relevant to Human Injuries? *Medical Hypotheses*, **81**, 747-748. https://doi.org/10.1016/j.mehy.2013.07.040

[6] Tator, C.H. (1995) Update on the Pathophysiology and Pathology of Acute Spinal Cord Injury. *Brain Pathology*, **5**, 407-413. https://doi.org/10.1111/j.1750-3639.1995.tb00619.x

[7] Osterholm, J.L. (1974) Noradrenergic Mediation of Traumatic Spinal Cord Autodestruction. *Life Sciences*, **14**, 1363-1384. https://doi.org/10.1016/0024-3205(74)90149-0

[8] Osterholm, J.L. and Mathews, G.J. (1972) Altered Norepinephrine Metabolism Following Experimental Spinal Cord Injury. Part 1: Relationship to Hemorrhagic Necrosis and Post-Wounding Neurological Deficits. *Journal of Neurosurgery*, **36**, 386-394. https://doi.org/10.3171/jns.1972.36.4.0386

[9] Schoultz, T.W. (1977) Microscopic Analysis of Early Histopathological Spinal Cord Alterations Following Trauma in Normal and Catecholamine-Depleted Cats. *Journal of the Neurological Sciences*, **32**, 283-295. https://doi.org/10.1016/0022-510x(77)90243-x

[10] Fayez, O., Simmons, H., Gwardjan, B., Nitin, S., Masood, F., Sledge, J., Abdullah, H. and Nesathurai, S. (2023) Correlation of Pathological Findings with MRI Imaging in Traumatic Spinal Cord Injury in Hyperacute Time Period in Non-Human Primate Model Pathological and radiological. *Open Journal of Veterinary Medicine*, **13**, 186-192.

[11] Fayez, O., Simmons, H., Gwardjan, B., Nitin, S., Masood, F., Sledge, J., Abdullah, H. and Nesathurai, S. (2019) Pathological and Radiological Features of Traumatic Spinal Cord Injury (Poster and Abstract). *Journal of Neuropathology & Experimental Neurology*, **78**, 579.

[12] Alderman, J.L., Osterholm, J.L., D'Amore, B.R. and Williams, H.D. (1980) Catecholamine Alterations Attending Spinal Cord Injury: A Reanalysis. *Neurosurgery*, **6**, 412-417. https://doi.org/10.1227/00006123-198004000-00009

[13] Rawe, S.E., Roth, R.H., Boadle-Biber, M. and Collins, W.F. (1977) Norepinephrine Levels in Experimental Spinal Cord Trauma. *Journal of Neurosurgery*, **46**, 342-349. https://doi.org/10.3171/jns.1977.46.3.0342

[14] Hedeman, L.S., Shellenberger, M.K. and Gordon, J.H. (1974) Studies in Experimental Spinal Cord Trauma. Part 1: Alterations in Catecholamine Levels. *Journal of Neurosurgery*, **40**, 37-43. https://doi.org/10.3171/jns.1974.40.1.0037

[15] Naftchi, N.E., Demeny, M., DeCrescito, V., Tomasula, J.J., Flamm, E.S. and Campbell, J.B. (1974) Biogenic Amine Concentrations in Traumatized Spinal Cords of Cats. *Journal of Neurosurgery*, **40**, 52-57. https://doi.org/10.3171/jns.1974.40.1.0052

[16] Eisenhofer, G. (2022) Monoamine-Producing Tumors. In: Rifai, N., Ed., *Tietz Textbook of Laboratory Medicine*, 7th Edition, Vol. 765, Saunders, 1-54.

[17] Bingham, W.G., Ruffolo, R. and Friedman, S.J. (1975) Catecholamine Levels in the Injured Spinal Cord of Monkeys. *Journal of Neurosurgery*, **42**, 174-178. https://doi.org/10.3171/jns.1975.42.2.0174

[18] Guttman, L. (1976) Disturbances of Vasomotor Control. In: *Spinal Cord Injuries*, Blackwell Publishing, 295-300.

[19] Schmid, A., Huonker, M., Stahl, F., Barturen, J., König, D., Heim, M., *et al.* (1998) Free Plasma Catecholamines in Spinal Cord Injured Persons with Different Injury Levels at Rest and during Exercise. *Journal of the Autonomic Nervous System*, **68**, 96-100.

https://doi.org/10.1016/s0165-1838(97)00127-6

[20] DeSantis, N. (2013) Autonomic Dysfunction. In: Nesathurai, S., Ed., *The Rehabilitation of People with Spinal Cord Injury*, 3rd Edition, Arbuckle Academic Publishers, 77-80.

[21] American College of Surgeons (2022) Neurogenic Shock and Systemic Pressure Related Therapy. Best Practice Guidelines: Spinal Cord Injury.

[22] Feloiu, F. and Dominguez, A. (2013) Clinical and Functional Evolution. In: Nesathurai, S., Ed., *The Rehabilitation of People with Spinal Cord Injury*, 3rd Edition, Arbuckle Academic Publishers, 32-36.