Neuronal Morphology in Rat Medial and Lateral Vestibular Nuclei after Blast Exposure

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Introduction

- Mild traumatic brain injury (mTBI) caused by blast exposure is a common cause of vestibular damage, particularly in military settings (Lien & Dickman, 2018; Long et al., 2009; Kabu et al., 2015).
- Vestibular damage can lead to debilitating symptoms such as dizziness, vertigo, and imbalance. The commissural inhibitory system between the bilateral vestibular nuclei plays a crucial role in vestibular compensation after such injuries (Baek et al., 2008; Dutia,2010).
- Understanding the structural changes in vestibular nuclei neurons following blast-induced mTBI is essential for developing effective rehabilitation strategies.

Objective

- The commissural inhibitory system between the bilateral vestibular nuclei plays a crucial role in vestibular compensation after unilateral damage.
- Here, the objective is to analyze the dendritic morphology of neurons in the rat medial vestibular nuclei (MVN) and lateral vestibular nuclei (LVN) using Golgi staining at 21 days after mild blast-induced traumatic brain injury (mTBI).

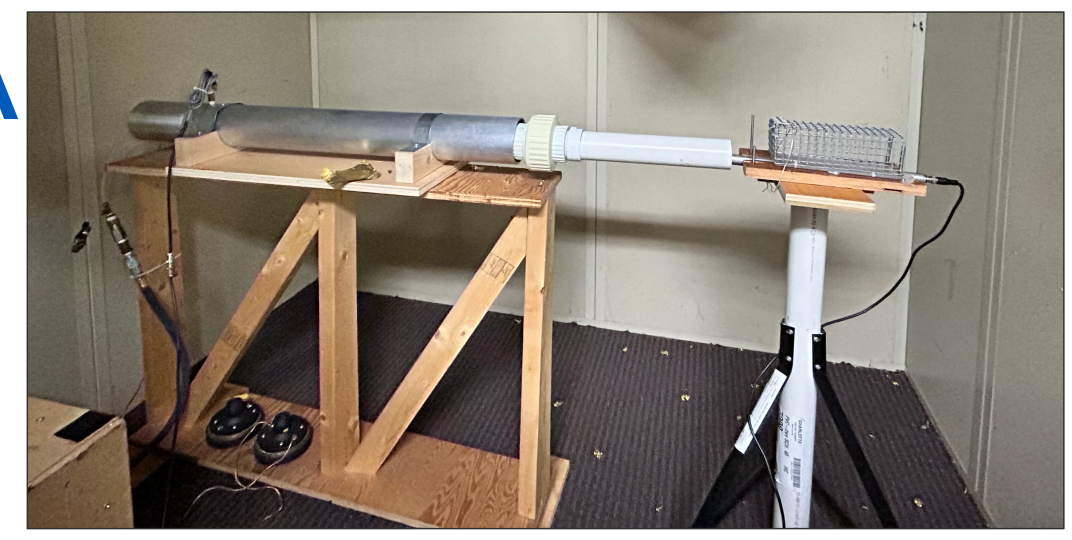
Methods

- MVN and LVN were collected for Golgi staining in sham control (n=3).
- The 21 days post-injury neurons were analyzed (n=3) from a blast-exposed rat (~180 dB SPL, 45 psi, <2ms duration).
- Sholl analysis was performed on stained neurons to quantify dendritic complexity using the Fiji software package. (SNT plugin)
- Images were preprocessed by inverting their color scheme.

Results

- The Sholl analysis plot shows sampled dendritic intersections as a function of distance from the neuronal soma.
- Sham controls exhibit a peak in intersections at ~60 µm from the soma.
- At 21 days post-injury, neurons display reduced dendritic length but an increase in complexity, with fewer intersections at most distances and a leftward shift in the peak.
- A 20th-degree fit of the sampled data closely matches the overall trends ($R^2 = 0.969$).

A



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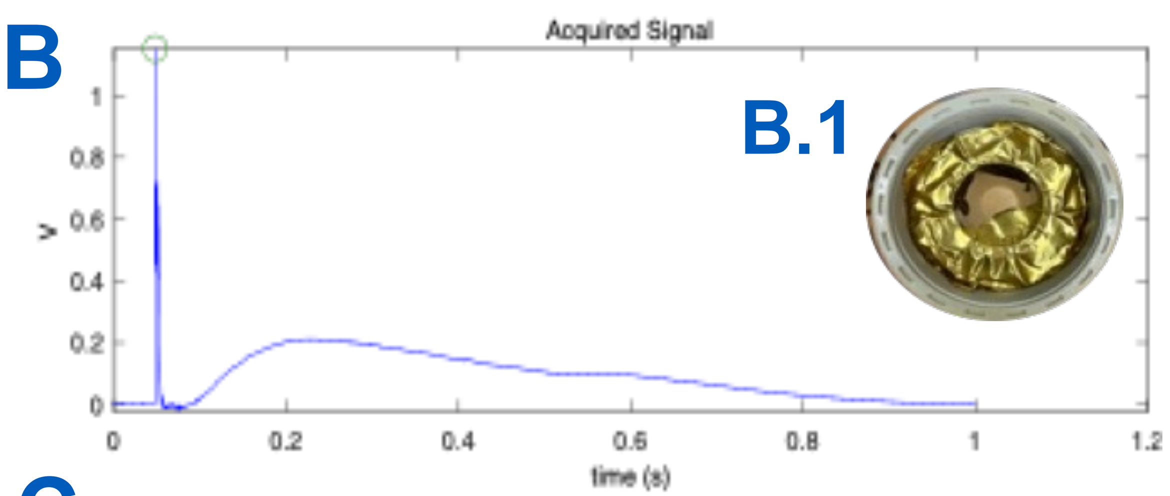

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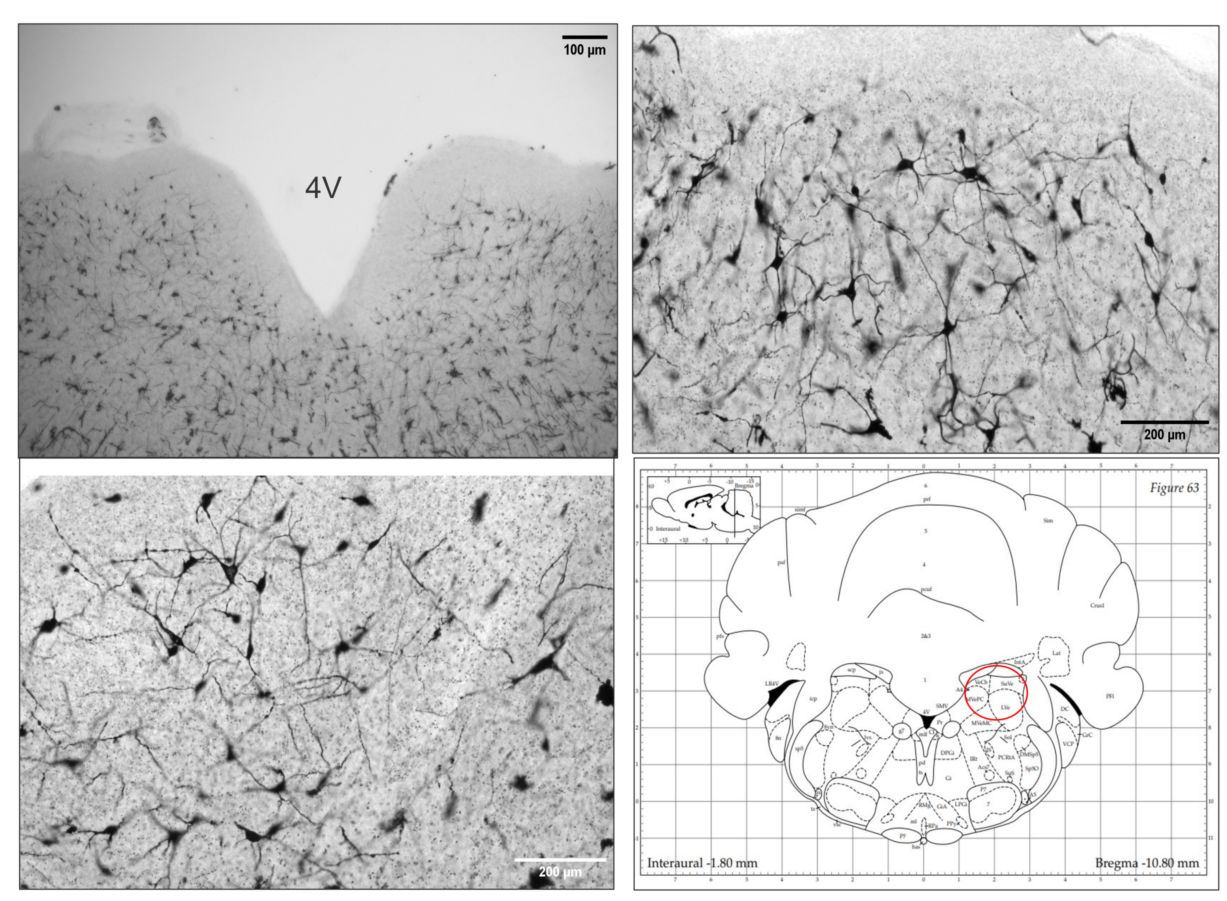
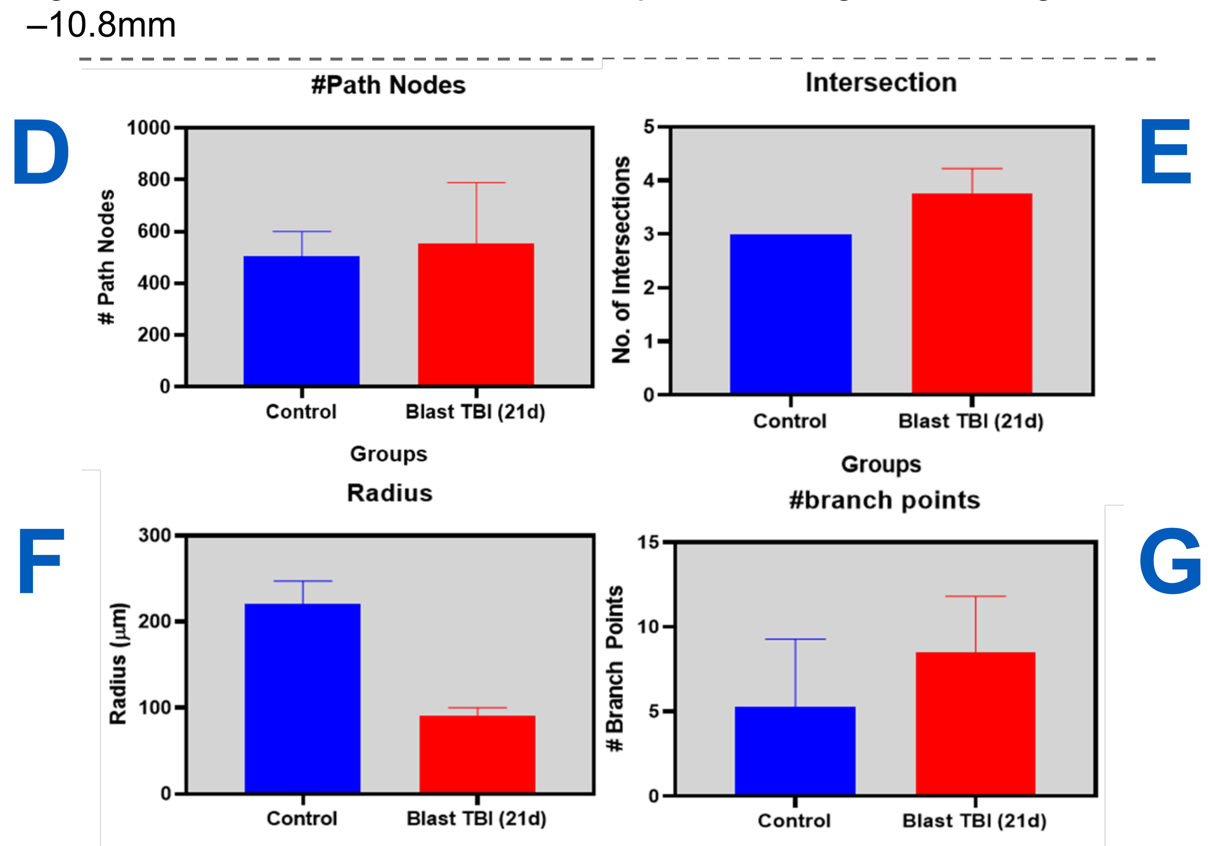


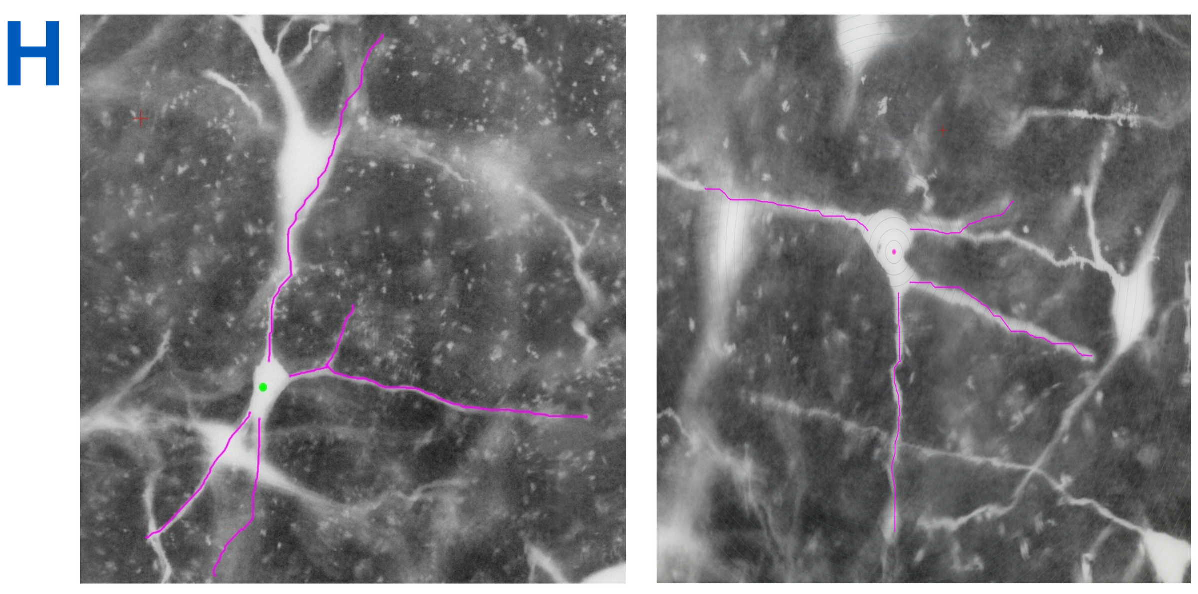
FigA. Schematic of positioning the blast tube, animal, and probe tip during animal blast wave exposures.
FigB. Impulse waveform (Blastwave) <2 t(s). **FigB1.** Brass after blast exposure.
FigC. MVN control 100 µm scale bar. Bregma level –10.80 mm. Upper right: MVN zoom. Lower left: LVN 200 µm. Lower right Atlas bregma level –10.8mm

D E F G

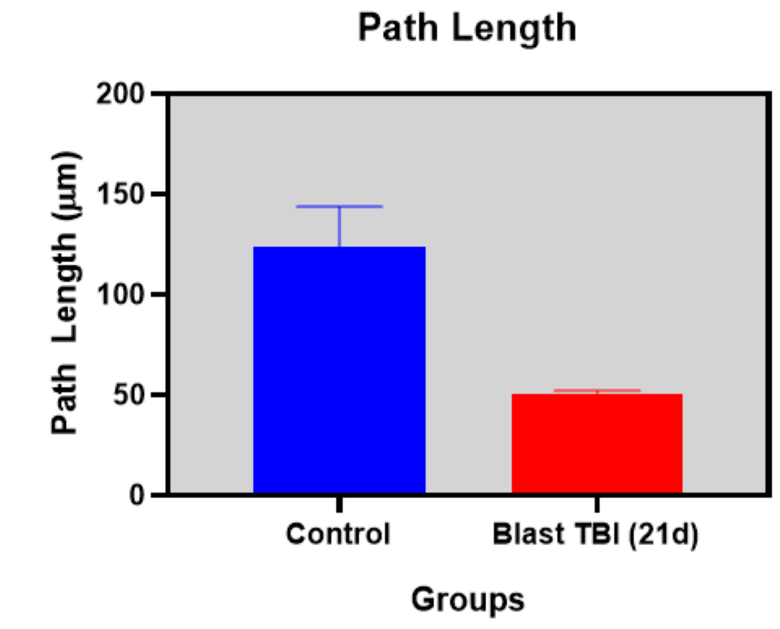


FigD. Number of Path Nodes: The mTBI group exhibits an increase in path nodes compared to the control, indicating modification of neuronal structure post-injury.
FigE. Number of Intersections: The mTBI group shows a marked increase in intersections, suggesting neuronal complexity and connectivity following blast exposure.
FigF. Radius: The mTBI group demonstrates a decreased neuronal radius, indicating potential shrinkage or atrophy of neurons in response to blast injury.
FigG. Number of Branch Points: The mTBI group significantly reduces branch points, implying a loss of dendritic arborization and simplified neuronal morphology post-injury.

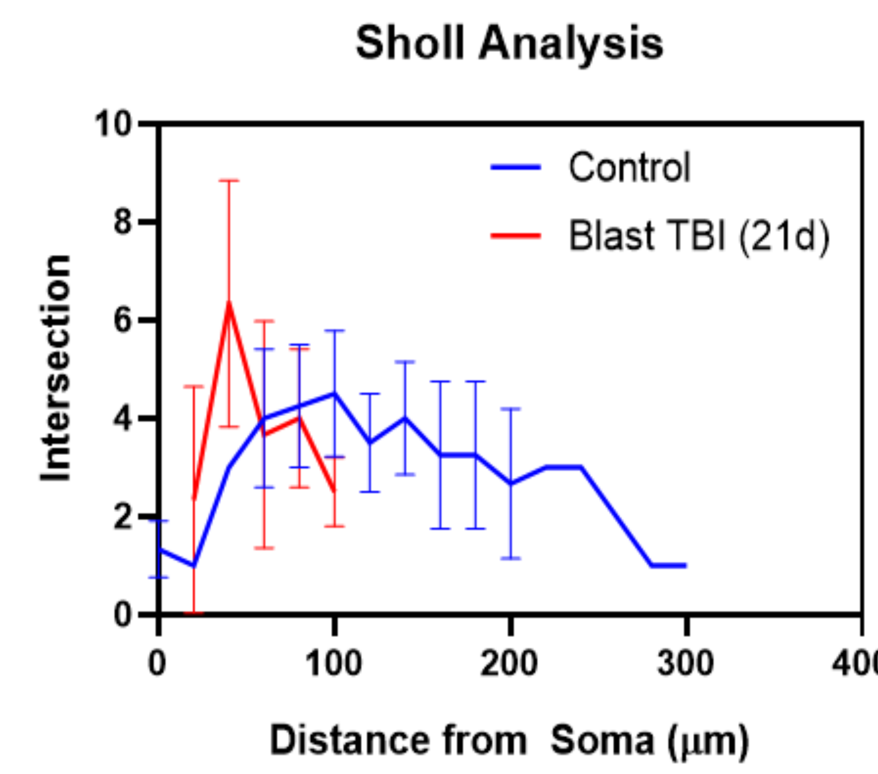
H



I



FigH. Inverted image in MVN. Segmentation process in Z plane.
FigI. Path length. The mTBI group exhibits a decrease in path length compared to the control group.



FigJ. Sholl analysis, intersection over the radius in the control group (blue line), and after 21 days of blast exposure. We can see more intersections in shorter distances after mTBI.

Conclusion

Blast exposure induces changes in MVN and LVN neuronal dendrite structure, with initial simplification followed by recovery.

These changes likely reflect plasticity in the commissural inhibitory system after vestibular damage.

Our Golgi staining approach may help elucidate vestibular rehabilitation and compensation mechanisms when describing this structural remodeling.

References

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