The Effects of Mechanical Traumatic Brain Injury on Bladder Function

Peter Zvara, MD, PhD1, Benjamin J. Moody1, Kalev Freeman, MD, PhD1, and Katarina Zvarova, MD, PhD1
1Department of Surgery University of Vermont

ABSTRACT

Hypothesis / aims of study
Approximately 1.5 million Americans sustain a traumatic brain injury (TBI) each year, with over 1 million seeking care in US emergency departments.[1] The initial transient benefit of elevated autonomic parameters following the acute TBI in 8-12% of patients shifts from protective to excessive resulting in paroxysmal or persistent sympathetic overactivity – dysautonomia.[2] Because of their complexity, micturition regulatory systems are particularly vulnerable to injury. Here, we sought to develop an experimental animal model for studying TBI-induced dysautonomia and associated bladder function/dysfunction.

Study design, materials and methods: A DSI dual-pressure recording device was implanted into the abdominal aorta and bladder dome to directly record bladder pressure (BP), arterial pressure (AP), and heart rate (HR). After a 7-day recovery period, fluid percussion brain injury was induced. Data were monitored continuously by telemetry for 24 hours before and immediately after injury, and then every day for 2 hours until post-trauma day 3. Correlations among neurological outcome, sympathetic parameters, and urodynamic parameters in the acute phase were evaluated.

Results
Pre-injury sympathetic and urodynamic parameters were within the physiological range (HR, 413.19 ± 18bpm; AP, 121±9mmHg; n=5); voiding frequency averaged 4/day and 9/night. Injury transiently increased maximum AP (158±41mmHg) and HR (449.43±18bpm). Mean time to 1st post injury micturition event was ~7±0.6 hours. Voiding frequency reduced to 2 voids/day and 4 voids/night and was associated with increase in voided volume. Frequency of voiding normalized within 2 days. However, the micturition pattern in subgroup of animals remained unstable with an increased incidence of non-voiding contractions.

Interpretation of results
We describe an experimental model for assessing autonomic and urodynamic responses to TBI and demonstrate the utility of a DSI telemetry system.

Concluding message
TBI induced transient neurogenic stress and changes in bladder behavior characteristic of bladder overactivity, suggest a role for TBI-induced dysautonomia in the etiology of bladder dysfunction.

MATERIALS AND METHODS

Figure 1. Fluid percussion head injury animal model: Animals were placed under general isoflurane anesthesia (2%) for surgical craniotomy with placement of a hollow, intracranial bolt. The bolt was introduced through the entire width of the cranium. While under anesthesia, this bolt was connected to a fluid percussion device, which was used to transmit a pendulum-driven fluid wave to the cerebral cortex, inducing a reproducible, non-lethal, neurologic injury. The bolt was then removed and the wound closed. Sham animals received anesthesia and scalp incision without the percussive injury. A 27-point neurological scoring system was used to assess neurologic outcome in first 60 minutes after the injury.

Figure 2. Radiotelemetry: Telemetry equipment (Data Sciences International (DSI) St. Paul, MN) interfaced with Dataquest™ A.R.T. software version 4.3 was used for the study. Rats were anesthetized with isoflurane (2-3%) delivered with 1% oxygen and surgically implanted with dual pressure HD-S21physio Tel® transmitters in the abdominal aorta and bladder. A midline laparotomy was performed to expose abdominal aorta and urinary bladder. One of the two pressure transmitters was introduced into the artery posterior to the vena cava; the other was implanted into the bladder dome for direct measurement of intravesical pressure. Body of the transmitter was placed intraabdominally and sutured to the abdominal wall. Rats were given 14 days to recover. Following recovery animals were challenged with TBI. The voiding and systemic pressure parameters recorded continuously during 24 hours before and after the injury were evaluated by averaging the result values from all micturition cycles. The voided volume parameters were recorded simultaneously by continuous scale readings.

RESULTS

Figure 3. Traumatic brain injury-induced changes in bladder parameters (upper left panel), heart rate (upper right panel), and systemic pressure (lower left panel), recorded by telemetry. The recordings represent raw data acquired 24 hours post injury. Note the prolonged time to 1st post injury micturition event, reduced voiding frequency with increased voided volume (lower left panel) and changes in bladder compliance (upper left panel), and voiding frequency following the brain injury.

Figure 4. Urodynamic profile of rat. Cystometrogram demonstrating one micturition cycle prior (left panel) and immediately after (right panel) the experimental brain injury. The low bladder compliance and impaired voiding pattern improved reaching normal values within 12 hours post injury. Note the significant increase in time required to trigger micturition following the injury.

Figure 5. Changes in basal (BP), threshold (TP), and micturition (MP) pressure (left panel), and arterial pressure (AP) and heart rate (HR) (right panel) before (gray columns) and 24 hours after (black columns) the brain injury.

CONCLUSIONS

1. Experimental TBI in rat is associated with transient dysautonomia.
2. TBI and/or associated neurogenic stress induces bladder dysfunction.
3. Nature of bladder dysfunction correlates with the neurologic outcome and/or severity of the injury.
4. Changes in bladder function that occur after the brain injury reflect the complex system innervation of lower urinary tract.
5. In rat TBI-induced changes affecting micturition reflex/urinary bladder occur mainly in the acute phase within the first 12 hours post injury.
6. DSI telemetry system is demonstrated to be a useful tool for continuous recording of multiple physiological parameters in an awake animal.

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