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The Effects of Mechanical Traumatic Brain Injury on Bladder Function

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±15bpm; AP, 121±9mmHg n=5); voiding frequency averaged 4/day and 9/night. Injury transiently increased maximum AP (158.4±21mmHg) 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.

References

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2. Baguley IJ, Heriseanu RE, Nott MT, Chapman J, Sandanam J: Dysautonomia after severe traumatic brain injury: Evidence of persisting overresponsiveness to afferent stimuli. American journal of physical medicine & rehabilitation / Association of Academic Physiatrists 2009;88:615-622.

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Were guidelines for care and use of laboratory animals followed or ethical committee approval obtained?	**Yes**
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