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

Specify source of funding or grant
Internal UVM Surgery Department fund

Is this a clinical trial?
No

What were the subjects in the study?
ANIMAL

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

Name of ethics committee
Institutional Animal Care and Use Committee of the University of Vermont. Animal care complied with Association for Assessment and Accreditation of Laboratory Animal Care and National Institutes of Health guidelines and was under the supervision of the University of Vermont’s Office of Animal Care (IACUC# 10-036).