Background: Maintaining adequate cerebral perfusion serves as a cornerstone in the medical management of patients with traumatic brain injury (TBI). Vasopressors (VP) are commonly utilized as an adjunct to achieve adequate perfusion pressures. However, the use of VP to improve clinical outcomes conflicts with the theory that excess catecholamine response in severe TBI patients leads to increased mortality. The impact of vasopressor utilization on the outcomes of TBI patients remains understudied. We sought to characterize the use of vasopressors in TBI patients and evaluate their impact on mortality. We hypothesized that vasopressor use would be associated with increased mortality.

Methods: A retrospective review was conducted of all TBI patients admitted to an ICU at an academic, urban, Level I trauma center from January 2014 to August 2016. Data collection included patient demographics, co-morbidities, injury characteristics, diagnoses, vasopressor use, and outcomes. The primary outcome was mortality. Patients who had any vasopressor administered (VP+) including dopamine, dobutamine, epinephrine, norepinephrine, phenylephrine, and vasopressin were compared to those who did not get any (VP-). A Cox regression model was utilized to adjust for differences between the two groups.

Results: There were 556 patients meeting inclusion criteria over the 32-month study period, of which, 83 (14.9%) received vasopressors. VP+ patients were younger (54.3 ± 21.1 vs. 59.7 ± 23.2 years, p=0.04) and had a significantly higher AIS head (4.4 ± 0.7 vs. 3.7 ± 0.6, p<0.01) compared to VP- patients. Bolt and/or intracranial pressure monitors were utilized significantly more often in VP+ patients (21.7% vs. 1.3%, p<0.01). In addition, these patients were more likely to develop either diabetes insipidus (2.4% vs. 0.0%, p=0.02) or cerebral salt wasting (3.6% vs. 0.0%, p<0.01). For VP+ patients, the most commonly utilized vasopressor was norepinephrine (75.9%), followed by phenylephrine (56.6%) and vasopressin (28.9%). The overall mortality was 9.2%, significantly higher in the VP+ cohort (42.2% vs. 3.4%, p<0.01). The use of each additional VP was associated with an increase in the mortality rate in a step wise fashion. After adjusting for confounding factors, VP+ patients had a significantly higher risk for in-hospital mortality (Adjusted Hazard Ratio: 2.77, adjusted p=0.01).

Conclusion: The use of vasopressors in the resuscitation of TBI patients is associated with a significantly higher mortality. Routine utilization of vasopressors to achieve adequate cerebral perfusion may not be justified. Further studies are required to explain the association between exogenous catecholamines and effect on mortality in this patient population.