The Effects of Traumatic Brain Injury on Bladder Function

Abstract

Traumatic brain injury (TBI) is a threat to proper neurological functioning in all individuals, and can result in symptoms such as incontinence or overactivity, problems that can severely decrease an individual’s quality of life. Unfortunately, the mechanisms behind this injury-induced bladder dysfunction are not well known, and so there are a limited number of effective treatments. This study seeks to characterize the relationship between injury-induced dyautonomia and bladder dysfunction in two ways, ultimately in the hopes of better understanding this dysfunction and producing more effective treatments in the future. In this experiment, systemic and bladder parameters will be simultaneously monitored, both before and after traumatic brain injury (TBI) is induced, to observe the physiological effects of TBI on bladder function. Further, tissue samples will be harvested four hours after injury, and immunohistochemistry (IHC) will be performed to determine injury-induced changes in adrenergic signaling and sympathetic activity. It is believed that experimentally-induced TBI will cause sympathetic hyperreflexia resulting in increased intermicturition intervals and volume retention, as well as changes in blood pressure and heart rate. Further, changes in the distribution of tyrosine hydroxylase, as well as beta-3 and alpha-1 adrenergic receptors are expected in tissue samples analyzed four hours post injury via IHC. We hope to discern trends in abnormal bladder function and associate them with changes in arterial pressure and heart rate, as well as structural changes within the bladder. This principal study of mechanical and physiological changes in the bladder is a crucial step towards more fully understanding injury induced bladder dysfunction, in the hopes of providing more effective treatments in the future.