Traumatic brain injury and sensory cortex: using barrel cortex to understand functional changes in the injured brain

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Traumatic brain injury (TBI), as occurs in car accidents or from falls, can result in persistent sensorimotor and cognitive deficits in humans. A likely major contributor to these deficits is altered sensory processing but there are few animal models of sensory processing in TBI. We investigated TBI-induced changes in sensory cortex neuronal responses, using the rat whisker tactile system and the cortex processing whisker-derived input.

TBI was induced using the weight-drop impact acceleration method which models the pattern of head and neck movements that occur in car accidents. We identified three factors likely to contribute to the brain injury: (a) the impact velocity/acceleration; (b) the relative motion of brain *vis-à-vis* the skull; and (c) whiplash neck motion. We initially characterized the impact of the first factor to determine how changes in this factor affected mortality and degree of behaviour deficits, with brain trauma created under inhalant isofluorane anaesthesia (3%). Then using a condition which caused significant TBI with low mortality we characterized the electrophysiological changes in sensory cortex underlying coding of simple and complex sensory input. Our results in terminal eectrophysiological experiments conducted under inhalant halothane anesthesia (~1.5%), indicate that within 24-48 hours post-TBI, there is a layer-dependent suppression of neuronal responsiveness coupled with tonic hyper-excitability in deep sensory cortical layers. However 8-10 weeks post-trauma, there is a significant hyper-responsiveness in the upper cortical layers with no changes in granular or infragranular cortical layers; there is also no longer any tonic hyper-excitation in any cortical layer.

These results suggest that immediately after trauma, the dominant effects appear likely related to shock wave or spreading depression effects that decrease with distance but that over time, cortical hyper-excitation to stimuli likely occurs through intra-cortical changes in the balance between excitation and inhibition. Given that the long-term changes occur only in cortical layers involved in intra-areal processing and long-range integration, these long-term changes in local inhibition must contribute to human sensorimotor deficits post-trauma.