due to hearing impairment. Histopathological evidence was overall mild, with presence of scattered blast intensity-dependent intracranial microhemorrhages. The most striking pathological feature was dose-dependent FD neurosilver and Fluorjade B staining of the superior colliculus, with some evidence of astroglia and microglia activation in the same areas. Molecular evaluations showed evidence of a mild increase in levels of spectrin breakdown products, suggestive of calpain activation, and of several cytokines both in frontal cortex and hippocampus. Levels of neuron specific enolase and brain-derived neurotrophic factor, were increased in plasma after blast exposure. Overall, a single blast exposure did not appear to cause more than mild evidence of brain injury in rat, with maximum impact on neurosensory structures and pathways.

Key words
primary blast, rat, TBI

D2-31

ROLE OF THE CONTRALESIONAL CORTEX IN FORELIMB RECOVERY AFTER EXPERIMENTAL TBI

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Deficits in sensorimotor function after clinical TBI are a significant problem with up to 30% of patients experiencing chronic impairment. Previously, we have shown that left-lateralized controlled cortical impact (CCI) injury in the rat sensorimotor cortex induces a trans-hemispheric shift in affected, right forelimb-evoked fMRI cortical activation over to the homotopic, contralesional cortex (CLCx). While spontaneous recovery of affected limb-use occurs in this model, it is unclear whether CLCX-shifted activation is an indicator of beneficial map plasticity or if this is maladaptive. In order to determine if the CLCx plays a functional role during post-injury recovery of limb-use, forelimb-reaching was tested before and during temporary silencing of the CLCx using intraparenchymal muscimol injection (versus vehicle, n = X/group) and this was performed longitudinally at 1 and 4 weeks after CCI injury in the adult rat. The results showed that at 1 week post-injury and before silencing, TBI-affected forelimb-reaching was reduced by 71% from pre-injury levels as expected from prior work ($P < 0.05$). Silencing the CLCx 1 day later resulted in deficits in the TBI-unaffected limb compared to pre-silencing levels ($P < 0.05$), simply indicating a correctly targeted cortical injection. In addition however, there was a complete reversal of the TBI-affected limb-reaching deficits to pre-injury levels ($P < 0.05$) indicating significant involvement of the CLCx in TBI-affected limb function acutely post-injury. By 4 wks post-injury, deficits in TBI-affected forelimb-reaching measured before a second period of silencing had spontaneously recovered to within 20% of pre-injury levels ($P > 0.05$). Although CLCx silencing one day later induced deficits in the TBI-unaffected limb as expected ($P < 0.01$), opposite to 1wk post-injury, TBI-affected-limb function was also significantly reduced to 32% of pre-injury levels (45% of pre-silencing, $P < 0.05$). This indicates that prior fMRI data showing an ipsilesional-to-CLCx-shift in activation likely underpins affected forelimb function chronically. The absence of any new ipsilesional regions of cortical fMRI activation during CLCx silencing further indicates the importance of the CLCx to affected limb function. Support: UCLA BIRC

Key words
cortical silencing, fMRI, neuroplasticity, staircase reaching task

D2-32

FINITE ELEMENT SIMULATION OF BRAIN DEFORMATION FROM SIX DEGREE OF FREEDOM ACCELERATION MEASUREMENTS OF MILD TRAUMATIC BRAIN INJURY

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Human mTBI biomechanics are complex and poorly understood, rendering screening efforts ineffective. Inertial rotation and translation are thought to cause diffuse brain trauma, but human tolerance to acceleration in all rotational and translational directions (six degrees of freedom, 6DOF) has not been measured for a human injury. Using novel instrumented mouthguards that rigidly couple to the upper dentition, we measured head collision biomechanics in full 6DOF, including the first complete measurements of human mTBI. Over 500 collisions among 31 subjects were measured at American football, boxing, and mixed martial arts events. Two subjects sustained a concussion during competitive play: one suffered loss of consciousness (LOC) while the other self-reported more subtle post-concussive symptoms, including headache, impaired concentration, and slowed reaction. Using the KTH finite element (FE) model, we mapped complex spatiotemporal kinematics measured in vivo onto the brain's anatomy. The LOC injury reported the highest principal strain (50%) among 50 randomly-selected non-injury collisions and the self-reported injury. Six non-injury collisions produced higher strains than the self-reported injury (18%), but in different anatomical regions. Maximum strain in both injuries occurred in the corpus callosum, and no non-injuries reached injury strain levels in this region. The LOC injury also predicted large strains in the brainstem. Our 6DOF measurement system predicted deformation in brain structures consistent with observed neurological deficits. Injury and non-injury collisions were distinguished by the severity and location of maximum tissue strain. Damage to the corpus callosum has been shown to disrupt interhemispheric communication and affect perception, while damage to the brainstem has been shown to induce LOC. While more data is required to characterize brain tissue mechanics across a wider spectrum of injuries, congruity between our measured kinematics, predicted tissue deformations, and observed symptoms indicates the promise of this system as a clinical tool.

Key words
biomechanics, finite element modeling, mild traumatic brain injury (mTBI), screening, sensors, sports concussion

D2-33

ANXIETY-LIKE BEHAVIOR IN MICE AFTER TRAUMATIC BRAIN INJURY: DISCUSSION AND COMPARISON OF COMMONLY-USED TESTS AND MEASUREMENTS

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Traumatic brain injury (TBI) survivors often exhibit long-term symptoms of anxiety, and anxiety-like behaviors in animal TBI models are a common functional measure. However, anxiety measures in experiments in brain-injured rodents have yielded variable results.