Increased endothelial calcium signals in cerebral vessels following traumatic brain injury

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Traumatic brain injury (TBI) can cause impairment in cerebrovascular function. Endothelial cell (EC) Ca^{2+} signals normally activate both nitric oxide (NO) and endothelium-derived hyperpolarizing factor (EDHF) vasodilatory pathways in cerebral arteries. Little is currently known about the impact of brain injury on vascular endothelial Ca^{2+} signaling. We studied the effects of TBI on EC and Ca^{2+} signals in rat basilar arteries. Arteries were harvested 24h after fluid percussion injury or sham surgery. We measured EC Ca^{2+} signals in slit-opened basilar arteries from TBI and control animals using the Ca^{2+} fluorophore Fluo2-leakage resistant and confocal microscopy. We found that localized EC Ca^{2+} signals were elevated in TBI animals. The optical waveforms of these events were consistent with two Ca^{2+} signaling modalities: 1) Ca^{2+} pulsars—mediated via endoplasmic reticulum Ca^{2+} release through inositol trisphosphate receptors and 2) Ca^{2+} sparklets—caused by Ca^{2+} entry through Ca^{2+}permeable channel (e.g. TRPV4 channels) in the EC plasma membrane. The frequency of both types of EC Ca^{2+} signaling events were elevated after TBI. These data suggest that altered EC Ca^{2+} signaling may play a role in abnormal cerebrovascular function after TBI.