Cortical spreading depolarizations (CSDs) are characterized by waves of diminished electroencephalography activity that propagate across the cortex with subsequent loss of ionic homeostasis. CSDs have been found in many pathological conditions, including migraine, traumatic brain injury, and ischemic stroke. Because of CSD-associated ionic and metabolic disturbances at the peri-infarct area after ischemic stroke, it is thought that CSDs exacerbate tissue infarction and worsen clinical outcomes. Microglia, the main innate immune cells in the brain, are among the first responders to brain tissue damage. Recent studies demonstrated that microglia play a critical role in Cortical spreading depolarization initiation and propagation [1)].

Current treatment guidelines to prevent delayed cerebral ischemia is limited to oral nimodipine, maintenance of euvolemia, induction of hypertension if ischemic signs occur and endovascular therapy for patients with continued ischemia after induced hypertension. Future investigations will involve agents targeting vasodilation, anticoagulation, inhibition of apoptosis pathways, free radical neutralization, suppression of cortical spreading depolarization and attenuation of inflammation [2)].

Cortical spreading depression (CSD) has been observed with relatively high frequency in the period following human brain injury, including traumatic brain injury and ischemic/hemorrhagic stroke. These events are characterized by loss of ionic gradients through massive cellular depolarization, neuronal dysfunction (depression of electrocorticographic [ECoG] activity) and slow spread (2-5 mm/min) across the cortical surface.

Previous data obtained in animals have suggested that even in the absence of underlying injury, neurosurgical manipulation can induce CSD and could potentially be a modifiable factor in neurosurgical injury.

Thirty-three of 37 patients with early focal brain injury (intracerebral hemorrhage and/or hypodensity) in contrast to 7 of 17 without displayed SDs during days 1-4 (sensitivity: 89% [95% confidence interval, CI: 75%-97%], specificity: 59% [CI: 33%-82%], positive predictive value: 83% [CI: 67%-93%], negative predictive value: 71% [CI: 42%-92%], Fisher exact test, $p < 0.001$). All 4 SD-related variables during days 1-4 significantly correlated with the volume of early focal brain injury (Spearman rank order correlations). A multiple ordinal regression analysis identified the PTDDD as the most important predictor.

The findings suggest that early focal brain injury after aSAH is associated with early SDs and further support the notion that SDs are a biomarker of focal brain lesions [3)].

Carlson et al., reported their initial experience with direct intraoperative ECoG monitoring for CSD.

They prospectively enrolled patients undergoing elective craniotomy for supratentorial lesions in cases in which the surgical procedure was expected to last > 2 hours. These patients were monitored for CSD from the time of dural opening through the time of dural closure, using a standard 1 × 6 platinum electrode coupled with an AC or full-spectrum DC amplifier. The data were processed using standard techniques to evaluate for slow potential changes coupled with suppression of high-

frequency ECoG propagating across the electrodes. Data were compared with CSD validated in previous intensive care unit (ICU) studies, to evaluate recording conditions most likely to permit CSD detection, and identify likely events during the course of neurosurgical procedures using standard criteria.

Eleven patients underwent ECoG monitoring during elective neurosurgical procedures. During the periods of monitoring, 2 definite CSDs were observed to occur in 1 patient and 8 suspicious events were detected in 4 patients. In other patients, either no events were observed or artifact limited interpretation of the data. The DC-coupled amplifier system represented an improvement in stability of data compared with AC-coupled systems. Compared with more widely used postoperative ICU monitoring, there were additional challenges with artifact from saturation during bipolar cautery as well as additional noise peaks detected.

CSD can occur during elective neurosurgical procedures even in brain regions distant from the immediate operative site. ECoG monitoring with a DC-coupled full-spectrum amplifier seemed to provide the most stable signal despite significant challenges to the operating room environment. CSD may be responsible for some cases of secondary surgical injury. Though further studies on outcome related to the occurrence of these events is needed, efforts to decrease the occurrence of CSD by modification of anesthetic regimen may represent a novel target for study to increase the safety of neurosurgical procedures [4].

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