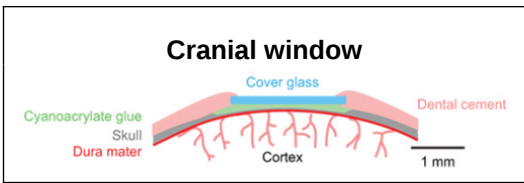
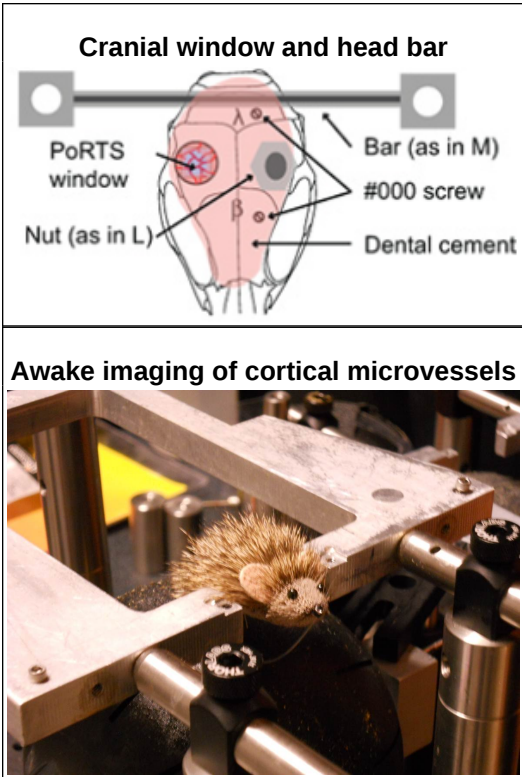


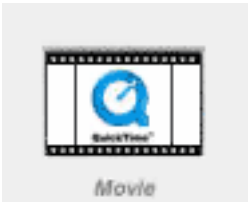
Introduction

A significant portion of the morbidity and mortality following aneurysmal subarachnoid hemorrhage (SAH) is due to delayed ischemic neurological deficits (DIND). Although large vessel vasospasm has been implicated as a cause of DIND, the presence of such spasm is not always correlated with DIND. We examined the cortical microvasculature as well as cerebral perfusion in awake mice after SAH in an experimental model typically associated with large vessel vasospasm.



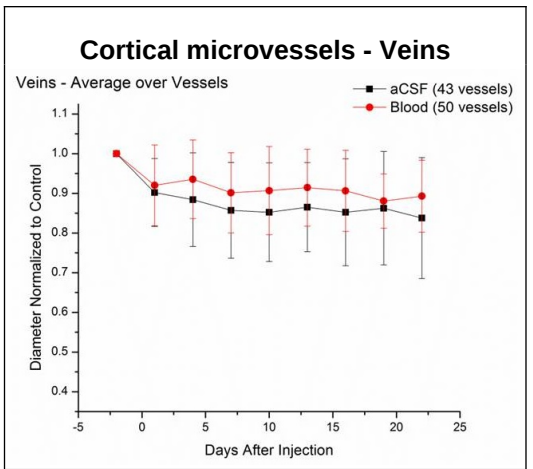
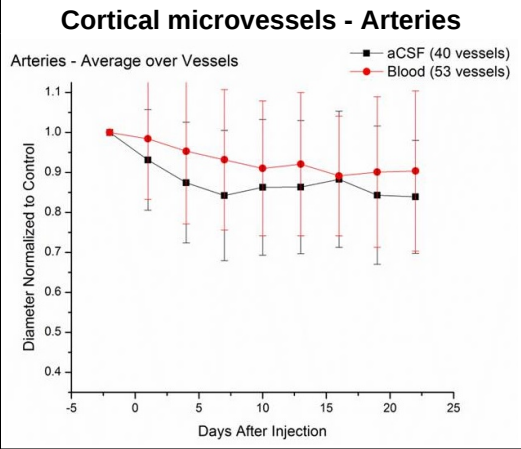
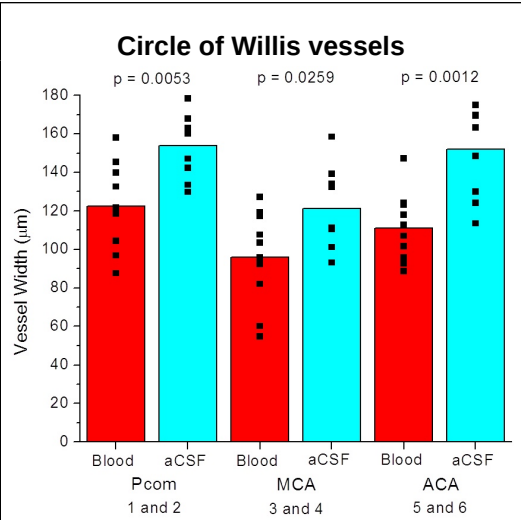
Methods

Twenty adult mice underwent cisterna magna (CM) injection of 60ul syngenic donor blood or artificial cerebrospinal fluid (aCSF). The mice were perfused at 72 hours, and Circle of Willis (COW) vessel diameters were measured. In a separate experiment, cranial windows were created in 10 mice that then underwent CM blood or aCSF injection. Cortical microvessels were measured in awake mice in the vasospasm period using two photon laser scanning microscopy. We also examined sensory-evoked increases in cerebral perfusion using laser Doppler and cerebral blood volume measurements.



Results

We observed a statistically significant difference in COW vessel diameter between the experimental and control groups at each of the sites measured (eg, MCA, $P=0.0259$; ACA, $P=0.0012$). In the second experiment, there were no significant differences in cortical microvessel diameter between the experimental and control groups. There were no differences in sensory-evoked increases in cerebral perfusion.



Conclusions

In a CM injection SAH model, we observed significant large vessel spasm but no cortical microvessel spasm or cerebral blood flow change. These results may have important implications for understanding the mechanisms of DIND.

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