

Rat Brain

Dai et al showed that [increased intracranial pressure](#) to 50 mm Hg in the healthy rat brain results in microvascular shunt flow characterized by tissue hypoxia, edema, and increased blood-brain barrier permeability. Dai et al now determined whether increased intracranial pressure results in neuronal injury by Fluoro-Jade stain and whether changes in cerebral blood flow and cerebral metabolic rate for oxygen suggest nonnutritive microvascular shunt flow.

Intracranial pressure was elevated by a reservoir of artificial cerebrospinal fluid connected to the cisterna magna. Arterial blood gases, cerebral arterial-venous oxygen content difference, and cerebral blood flow by MRI were measured. Fluoro-Jade stain neurons were counted in histologic sections of the right and left dorsal and lateral cortices and hippocampus.

Arterial pressure support if needed by IV dopamine infusion and base deficit corrected by sodium bicarbonate.

Fluoro-Jade stain neurons increased 2.5- and 5.5-fold at intracranial pressures of 30 and 50 mm Hg and cerebral perfusion pressures of 57 ± 4 (mean \pm SEM) and 47 ± 6 mm Hg, respectively ($p < 0.001$) (highest in the right and left cortices). Voxel frequency histograms of cerebral blood flow showed a pattern consistent with microvascular shunt flow by dispersion to higher cerebral blood flow at high intracranial pressure and decreased cerebral metabolic rate for oxygen.

High intracranial pressure likely caused neuronal injury because of a transition from normal capillary flow to nonnutritive microvascular shunt flow resulting in tissue hypoxia and edema, and it is manifest by a reduction in the cerebral metabolic rate for oxygen ¹⁾.

¹⁾

Dai X, Bragina O, Zhang T, Yang Y, Rao GR, Bragin DE, Statom G, Nemoto EM. High Intracranial Pressure Induced Injury in the Healthy Rat Brain. Crit Care Med. 2016 Mar 11. [Epub ahead of print] PubMed PMID: 26974548.

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