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Extracorporeal Blood Shunt Mimicking Aneurysm Rupture: New Rabbit Subarachnoid Hemorrhage Model for the Study of Delayed Cerebral Vasospasm.

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Introduction: Due to relatively low costs and easy handling rabbits became the most popular species in cerebral vasospasm research. The most often applied technique to simulate subarachnoid hemorrhage (SAH) in the rabbit is injection of blood into the cisterna magna. This technique comprises examiner-dependent variables and does not closely represent the human pathophysiological sequelae of ruptured cerebral aneurysm. The degree of vasospasm produced in this model is mild. That contributes to the fact that numerous therapeutic procedures appear to relieve experimentally induced vasospasm but turn out to be ineffective when used clinically.
Methods: Adult female New Zealand rabbits were assigned to two groups (SAH group, n=10 and controls, n=2). SAH was performed by shunting blood from the subclavian artery into the great cerebral cistern. An intermediary flow meter measured the blood volume which streamed under arterial pressure into the subarachnoid space. Intracranial pressure (ICP), arterial blood pressure, heart rate, arterial blood gas status, and neurological status were monitored throughout the experiments. The magnitude of spasm in the basilar artery was determined by comparison of pre-SAH (day 0) and post-SAH (day 3) angiograms and post-mortem morphometric analysis of the basilar artery as well as gross examination of the brain.
Results: A total of 18 experiments and 36 angiograms were performed. ICP (42.6 ± 1.2 mmHg) rose to diastolic blood pressure (39.7 ± 12.3 mmHg) in 97 ± 35 seconds and fall to a steady state within 185 ± 73 seconds. SAH induced vasoconstriction of the basilar artery was 52.7 ± 8.4%. Coronal sections of basilar arteries at proximal and middle brainstem level demonstrated significant vasoconstriction on day 3 after SAH induction with massive corrugation of elastic internal lamina. Post-mortem gross examination of the brain showed large blood clots located around the brainstem and ventral surface of the brain.
Conclusions: This novel technique of SAH induction resulted in significantly higher degree of delayed cerebral vasospasm compared to all other previous reported rabbit models. The severity of vasospasm attained offers a unique opportunity to evaluate future therapeutic treatment options. Monitored physiological parameters confirmed the close relation to the human situation of intracranial aneurysm rupture. Exact time course of the new model and its role to study early brain injury after SAH remains to be determined.

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