

Rat Model

see [Sprague Dawley rat](#)

see [Wistar rat](#).

see [Rat Brain](#).

Rat Model for Practicing Microsurgical [Vascular Anastomosis](#) ¹⁾.

[Cerebral vasospasm](#) as a delayed, possibly treatable sequel of [subarachnoid hemorrhage](#) (SAH) is a focus of [experimental animal research](#). For this purpose, the rat is not a good [model](#) because of the difficulty creating a stable subarachnoid clot that persists > 1 to 2 days and could induce [vasospasm](#). Only in rat models with a high [mortality](#) of ~ 50% or more can [SAH](#) and its effects be investigated. Therefore, other animals than [rodents](#) are used for investigating the delayed effects of SAH. Only animal studies addressing the acute effects of SAH use rats.

Ehlert et al. from the Department of Neurosurgery, Asklepios Klinik St. Georg [Hamburg](#), Asklepios Klinik Altona, Hamburg, University of Giessen, Universitätsklinikum Schleswig-Holstein, Campus Lübeck, Universitätsklinikum Hamburg, Germany, designed a model that allows intensive [clot](#) formation combined with low [mortality](#) to facilitate studies on the delayed effects of experimental SAH, for example, delayed [vasospasm](#) or other alterations of [vessels](#).

After [in vitro](#) acceleration of the clotting process in the rats blood by tissue factor and preliminary in vivo testing, we induced a SAH by injecting blood together with tissue factor in 22 rats. They analyzed clot expansion, length of clot persistence, chronic alterations, and histologic changes.

The injection of blood supplemented by tissue factor led to persistent voluminous blood clots in the [subarachnoid space](#) close to the large arteries. Despite the pronounced SAH, all animals survived, allowing investigation of delayed SAH effects. All animals killed within the first 7 days after surgery had extensive clots; in some animals, the clots remained until postoperative day 12. During further clot degradation connective tissue appeared, possibly as a precursor of SAH-related late hydrocephalus.

The injection of blood together with tissue factor significantly improves SAH induction in the rat model. This rat model allows studying delayed SAH effects as found in humans ²⁾.

Spontaneously hypertensive rat in Neurosurgery

- [Determinants of Outcome After Endovascular Middle Cerebral Artery Occlusion in Rats in the SPAN Trial](#)
- [Superior cervical ganglionectomy attenuates vascular remodeling in spontaneously hypertensive rats](#)
- [Effects of bilateral renal denervation on open-loop baroreflex function and urine excretion in spontaneously hypertensive rats](#)
- [Renal denervation improves mitochondrial oxidative stress and cardiac hypertrophy through](#)

[inactivating SP1/BACH1-PACS2 signaling](#)

- [Renal denervation alleviates vascular remodeling in spontaneously hypertensive rats by regulating perivascular adipose tissue](#)
 - [Afferent Renal Denervation Attenuates Sympathetic Overactivation From the Paraventricular Nucleus in Spontaneously Hypertensive Rats](#)
 - [The effects of renal denervation on blood pressure, cardiac hypertrophy, and sympathetic activity during the established phase of hypertension in spontaneously hypertensive rats](#)
 - [Methylglyoxal accumulation contributes to accelerated brain aging in spontaneously hypertensive rats](#)
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Spontaneously hypertensive rats (SHR) are a commonly used animal model for studying hypertension (high blood pressure) and related cardiovascular conditions. These rats were selectively bred for their propensity to develop hypertension, making them a valuable tool for research in this field.

SHR are known to develop high blood pressure as they age, resembling certain aspects of human essential hypertension. They exhibit characteristics such as elevated systolic and diastolic blood pressure, increased vascular resistance, and organ damage associated with hypertension, including cardiovascular and renal abnormalities.

Due to their genetic predisposition to hypertension, SHR have been extensively utilized in various studies to investigate the pathophysiology, underlying mechanisms, and potential treatments for hypertension. These rats have contributed significantly to our understanding of the complex factors involved in the development and progression of hypertension, as well as the impact of hypertension on target organs.

Researchers often use SHR to study the effectiveness of antihypertensive drugs, explore the relationship between hypertension and other diseases, investigate genetic factors influencing blood pressure regulation, and examine the physiological and molecular mechanisms involved in hypertension-related complications.

It's worth noting that while SHR are a valuable animal model for studying hypertension, findings from animal studies may not always directly translate to human conditions. Nonetheless, SHR remains a widely used tool in preclinical research related to hypertension and cardiovascular health.

Lolansen et al. aimed to elucidate the [molecular mechanisms](#) underlying the [hydrocephalus etiology](#) in spontaneously hypertensive rats (SHRs), which develop [communicating hydrocephalus](#) without the need for surgical induction.

Magnetic resonance imaging was employed to delineate brain and CSF volumes in SHRs and control Wistar-Kyoto (WKY) rats. Brain water content was determined from wet and dry brain weights. CSF dynamics related to hydrocephalus formation in SHRs were explored in vivo by quantifying CSF production rates, ICP, and CSF outflow resistance. Associated choroid plexus alterations were elucidated with immunofluorescence, western blotting, and through use of an ex vivo radio-isotope flux assay.

SHRs displayed brain water accumulation and enlarged lateral ventricles, in part compensated for by a smaller brain volume. The SHR choroid plexus demonstrated increased phosphorylation of the

Na⁺/K⁺/2Cl⁻ cotransporter NKCC1, a key contributor to choroid plexus CSF secretion. However, neither CSF production rate, ICP, nor CSF outflow resistance appeared elevated in SHRs when compared to WKY rats.

Hydrocephalus [development](#) in SHRs does not associate with elevated ICP and does not require increased CSF secretion or inefficient CSF drainage. SHR hydrocephalus thus represents a [type](#) of [hydrocephalus](#) that is not life threatening and that occurs by unknown disturbances to the [cerebrospinal fluid dynamics](#) ³⁾.

¹⁾

Tayebi Meybodi A. Rat Model for Practicing Microsurgical Vascular Anastomosis. J Reconstr Microsurg. 2019;35(7):e6. doi:10.1055/s-0040-1715645

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Ehlert A, Budde U, Middendorff R, Manthei G, Kemmling A, Tiemann B. Reintroduction of the Rat for Experimental Subarachnoid Hemorrhage with Accelerated Clot Formation: A Low Mortality Model with Persistent Clots as a Precondition for Studies in Vasospasm. J Neurol Surg A Cent Eur Neurosurg. 2018 Sep;79(5):424-433. doi: 10.1055/s-0038-1641561. Epub 2018 Jul 4. PubMed PMID: 29972859.

³⁾

Lolansen SD, Barbuskaite D, Ye F, Xiang J, Keep RF, MacAulay N. Spontaneously hypertensive rats can become hydrocephalic despite undisturbed secretion and drainage of cerebrospinal fluid. Fluids Barriers CNS. 2023 Jul 4;20(1):53. doi: 10.1186/s12987-023-00448-x. PMID: 37403103.

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