Description for the general public

Stroke is the main health problem in the world. Most cases of stroke are caused by the blockage of blood flow in the arteries of the brain. The last decades have brought tremendous advances in the treatment of stroke with interventional neuroradiology methods that allow the removal of the clot and the restoration of blood flow in the brain, but such therapies must be applied within the first hours of the stroke. Recently, significant discoveries have been made in the field of molecular and cellular mechanisms of secondary brain damage in ischemic stroke, mainly based on rodent studies. Many clinical trials based on results from rodent experiments failed due to differences between the small lissencephalic mouse brain and the human brain. Our group has recently developed a stroke model based on a minimally invasive endovascular technique. We are able to provide a embolus-inducing solution that is labeled with a magnetic resonance contrast agent (MRI) to induce clot formation in the arteries of the brain and monitor this process with MRI. Our pilot study indicates that we can induce changes similar to those that occur in stroke patients. In addition, the damage resulted in clear neurological deficits in the contralateral limbs manifested primarily by severe paresis of the hindlimbs, however, while being ambulatory and without the need for intensive care.
The main goal of this project is to investigate whether it is possible to develop a method of treating patients after a longer period of cerebral ischemia than the currently recommended 4-6 hours. We suggest using our new model of ischemia in domestic pig as well as using a typical rat stroke model to characterize a few basic issues, including the assessment of reperfusion consequences initiated 6 or 10 hours after ischemia, characterization of ischemic inflammatory processes, determination of a reliable quantitative motor deficit assessment in pigs after induction of stroke as well as evaluation of the usefulness of immunomodulatory treatment by blocking the IL-17 and MMP-9 molecular pathways.