Sleep apnea syndrome (SAS) is a disease characterized by an air-flow limitation or obstruction in the airway tract. This condition is often correlated with snoring. SAS often manifests as arousals during sleep, nocturia, chronic tiredness, morning headaches, daytime sleepiness. It is responsible for autonomic nervous system stimulation, inflammatory response, oxidative stress, impaired glucose and fat metabolism and hormone dysregulation. Described mechanisms lead to several pathological conditions, such as arterial hypertension, cardiovascular disease, psychological abnormalities. Stress generated by frequent arousals stimulates immunological system, inflammatory process, leading to atherosclerosis. SAS is an independent risk factor for ischemic stroke. The correlation between SAS, endothelium dysfunction and atherosclerosis was described in published epidemiological studies. Endothelial function improvement after treatment with continuous positive airway pressure (CPAP) was also observed. The pathway from chronic hypoxia to immunological response and vascular endothelium damage has not yet been fully understood. The aim of the study is to prove a hypothesis of a negative influence of SAS on atherosclerotic plaque stability (increased risk of its rupture) and to determine whether SAS worsens post-operative course in patient treated for carotid artery stenosis. Previous research (including our own unpublished data) suggest frequent occurrence of sleep apnea in population with carotid artery stenosis (CAS) . Still there is no data about SAS- related complications of surgically treated carotid artery stenosis and about influence of surgery on SAS severity.

Sleep apnea syndrome is diagnosed with polysomnography (PSG) and polygraphy (PG). PG is performed overnight and includes pulseoximetry, airflow, respiratory muscles movement (thorax and abdomen) recording. In our project a polygraphy (PG) will be used. The test enables us to describe breathing disorders during sleep.

Patients with atherosclerotic carotid artery stenosis, who require surgical treatment (both endarterectomy and stent implantation) will be enrolled into the project. After overnight diagnostic test (PG) blood samples will be drawn for laboratory tests. Inflammatory and angiogenic factors as well as metabolomic profile will be assayed. Additionally atherosclerotic plaque removed during carotid surgery will be assessed for plaque instability (histopathological and immunochemical examination).

Study hypothesis assumes that SAS is correlated with atherosclerotic plaque instability, caused by inflammatory response to chronic intermittent hypoxia, hypoxia induced gene overexpression and increased activity of proteolytic enzymes. All above lead to higher risk of sudden vascular incidents (brain stroke, transient ischemic attack). Inflammatory markers, hypoxia induced factors and angiogenic factors will be elevated in patients with SAS. We also plan to assess if carotid surgery has an effect on SAS severity. Special importance of research results is expected among patients with asymptomatic carotid artery stenosis. Therapeutic dilemma among that group is often noticed. Still there is no predictive factor describing patients requiring surgical treatment of CAS. Sleep apnea might be a valuable one.