Background: Glycocalyx represents a protective cellular coat on a sugar basis. It serves as a communication medium with outside environment. Glykokalyx also covers the inner apical surface of endothelial cells where it is called the endothelial glycocalyx (EG). Research from last decade declare its pivotal role in physiology and pathophysiology of microcirculation. EG is prone to be damaged in critical conditions but there are more questions than what we actually know about this complex structure. Also, there are new methods being developed for more precise description of EG condition.

The aim: The aim of this thesis is to evaluate the level of contemporary evidence about EG and to evaluate methods of its assessment. To describe the condition of EG in experimental model of cardiac arrest (CA) in pig and in experimental model of iatrogenic hypernatremia in rabbit. To describe the influence of different types of anesthesia on EG in patients in perioperative care. To describe the dynamic changes of EG in patients in critical care. To evaluate response of EG to fluid challenge of 500 ml of normal saline in healthy volunteers and the response of EG to administration of lipid emulsion in patients in critical care. To describe the condition of EG in patients with dyslipidemia, with long term home parenteral nutrition and to describe the condition of EG in healthy young individuals. The thesis was also aimed at identification of protective agents and approaches to EG and to suggest some future directions of the research.

Methods: EG was assessed by using two methods in experiment and in clinical studies. First by video microscopical investigation of the sublingual microcirculation by a specialized camera based on Side–stream Dark Field imaging technology and by automatic assessment of the acquired recordings using a specialized software GlycoCheck. This software provides a Perfused Boundary Region parameter (PBR) which indirectly reflects the height of the EG. And second by concentration of EG degradation products: syndecan–1 and syndecan–4 assessed by Enzyme Linked Immunosorbent Assay.

Results: PBR did not raised significantly in the porcine model of CA not even in 20 minutes after the return of spontaneous circulation. In experimental model of iatrogenic hypernatremia in rabbit the hypernatremia led to the significant raise on the PBR but only to insignificant elevation of syndecan–1 concentration. In clinical studies, fluid challenge by 500 ml of normal saline led to insignificant increase in PBR value in healthy volunteers. The PBR was significantly increased in patients after hip or knee replacement surgery in both general and neuraxial anesthesia. In general anesthesia the increase in PBR was significantly higher than in neuraxial anesthesia two hours after the procedure. In critical care PBR was significantly higher in patients in septic shock in patients with insidious onset of the critical condition and in patient on continuous renal replacement therapy. Administration of lipid emulsion in patients in surgical critical care led to non–significant increase in PBR value and to a significant decrease in syndecan–1 and syndecan–4 levels. In patients with home long term parenteral nutrition the PBR parameter and concentration of syndecan–1 were significantly higher than in patients with dyslipidemia. Concentration of syndecan–4 was unaffected. In the end the thesis we made the investigation in healthy young individuals to obtain reference values of PBR parameter and syndecan–1 concentration. We also identified protective agents and approaches for EG and suggested future areas of EG research.

Conclusion: There is still no method to evaluate conclusively the EG in a clinical frame. Based upon our results we can see that EG can be altered in our clinical practice by inadequate fluid therapy, by the type of the anesthesia and when continuous renal replacement therapy is required. Lipid emulsion seemed to have a protective effect on EG.