Insulin resistance is one of hot topics of present medicine not only in its braches focused on civilization diseases such as diabetology or obesitology but also in other fields, namely in intensive care medicine. Hyperglycemia caused by accelerated insulin resistance is observed in critically ill patients very often. It has many undesirable effects and potential complications. This phenomenon is caused by a network of different mechanisms, especially by systemic inflammatory response and subsequent burst of stress hormones and proinflammatory cytokines which directly participate in the ethiopatogenesis of insulin resistance. Recent investigations documented that many of proinflammatory cytokines are directly produced by adipose tissue. Although basic mechanisms of insulin resistance and type 2 type diabetes mellitus in obese subjects are somewhat different in both pathologie overproduction of some proinflammatory cytokines by adipose tissue may play a role. It has been demonstrated that correction of hyperglycemia in criticially ill patient by intensive insulin therapy leads to reduction of thein morbidity and mortality. Tight glycemic control requires a frequent blood glukose measueremnts increasing the workload of ICU personnel Therefore, the development of systems for continuous measuring of glycemia and effective automated dosage of insulin has been started.

The aim of our work was the evaluation of possible role of subcutaneous and visceral (epicardial) adipose tissue production of proinflammatory cytokines on the development of insulin resistance in critically ill patiens. We also aimed at testing the protocols to normalize blood glukose levels in these patients. Another aim of our research was to investigate the subcutaneous adipose tissue, as an alternative site for continuous measurement of glycemia using microdialysis method.

Patients hospitalized on postoperative intensive care unit of 2nd Department of Cardiovascular surgery, 1st Faculty of Medicine, General University Hospital in Prague who underwent elective cardiac surgery and required minimally short-term resuscitation care were included into our studies. The studies were performed during perioperative period.

We documented that cardiac surgery operation increased both serum and mRNA levels of proinflammatory cytokines in both subcutaneous and epicardial adipose tissue. This elevation was accompenied by a development of insulin resistance as measured by increased insulin requirements for establishment of euglycemia.

We conclude that endocrine dysfunction of adipose tissue plays an important role in the development of insulin resistence in critically ill patients. Therapeutic suppression of proinflammatory cytokines production in fat may in turn lead to partial attenuation undesirable insulin resistance and hyperglycemia of critically ill patients.

We also demonstrated good correlation between arterial and interstitial concentrations of glucose in critically ill patients and satisfactory effectiveness of computer algorithm in tight glycemic control of these patients. These results also open the possibility of the development of automated systems combining continuous glucose monitoring with continuous insulin administration.