Patients with a brain injury must overcome a central neurological insult and a systematic metabolic response. The systematic response includes hypermetabolism, hypercatabolism, altered vascular permeability, increased hormone and cytokine release, altered gastric emptying, altered mineral metabolism and altered immune status. This response may initiate mechanisms which lead to secondary brain injury and may adversely affect the function of other organs. Last two decades have improved our knowledge of pathophysiology in patients with primary and secondary brain injury including traumatic contusions. In a moment of injury, primary brain injury which is traumatic contusion can not be therapeutically prevented it can only have a preventive effect. Secondary brain injury can be therapeutically affected although both types of injuries have common pathophysiological signs of disturbed metabolism on cells and subcellular level. Research methods based on molecular biology opens a new zone of research reaction of central neurological system for trauma or ischemia with effort to get involved immediately at the beginning of developing adverse pathophysiological cascade leading to a death of neurotic cell. The brain contusion is a primary centre of brain injury with anatomic changes which can be graphically proved. In the course of multinumerous or large contusions, a dynamic progress is gradually leading to global symptoms with decline of consciousness on the strength of progression of cerebral edema and dynamic changes in contusion centre. While in its centre ischemia can lead to necrotic tissues in the near surrounding, in pericontusional zone (penumba) causes disorder of cerebral blood flow of starting apoptosis cascade leading to a death of brain cell. A connection between intracranial cerebral pressure (ICP), cerebral perfusion pressure (CPP) and cerebral blood flow (CBF) is integrated and dynamic. In principle a supply of oxygen and energetic substrate on the way through blood stream is dropping by increasing pressure in the skull.
Understanding of pathophysiological circumstances is supporting component of diagnostical and medical process. Cerebral multimodal monitoring of patient in departments of intensive care is focused on monitoring of dynamic parameters indicating secondary brain injury. In return it enables to optimise medical procedure and some parameters may be used for predictions of treatment results. An extensive brain contusion with perifocal or difussed oedema is accompanied with increase of intracranial cerebral pressure, brain ischemia, hypoxia, lactate congestion, ion disbalance, stress response which includes hyperglycemia. 51 patients with brain contusion were monitored having consciousness disorder on their entry in a range of GCS 3-8. These patients were treated in departments of intensive care in Department of Neurosurgery or Anaesthesiology, Charles University Hospital in Pilsen in years 2000-2005. All patients were monitored, made sedation, relaxation and were ventilated. In all patients intracranial cerebral pressure was monitored and levels of osmolality, glycemia, lactate, sodium and potassium were also followed. A dynamic range of these changes was correllated with clinical result of treatement – Glasgow Outcome Scale (GOS). A favourable neurological result (GOS 4-5) was reached in 28 patients, an unfavourable in 23 patients, 8 of them died. After 6 months in 43 patients who survived final average of GCS was 11, Karnovsky score was 61,2. In patients with favourable GOS, value of ICP reached lower levels, the same as entry and final levels of glycemia and lactate than in the patients with unfavourable neurological result. The entry level of lactate in the patients with unfavourable GOS substaintially exceeded normal span and became significant, independent and prognostic factor of poor prognosis. On the other hand final lactate in unfavourable GOS was in physiological span and did not have any prognostic importance. In the start the patients who died had significant initial hyponatremia, intracranial hypertension, hyperglycemia and permanent hyperosmolality. The factors statistically endorsing unsatisfactory result of GOS with intracranial pressure are: age of patients above 60 years - having 25 times higher risk of reaching result of GOS 1-3, patients with S-Na >142 mmol/l projective factor of GOS 1-3 and osmolality >301 mosmol/l with 6 times higher risk of GOS 1-3. The most significant unfavourable prognostic impact had actual presence of original intracranial pressure in combination with hyperglycemia, hyperlactemia and hyperosmolality. Together they introduce indicators of brain injuries degree and they predict poor therapeutic result in these patients.