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Multiparametrische Charakterisierung der perihämorrhagischen Zone nach ipsi- und kontralateraler intrazerebraler Blutung im Schwein

Promotionsfach: Neurochirurgie
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The very early pathophysiologic changes occurring during the first hours after intracerebral hemorrhage (ICH) in humans are not well understood. In this thesis we have presented the first experimental multi-parametric neuromonitoring ICH model in swine.

In small volume ICH (1.5ml) we have demonstrated an elevated ICP correlated with decreased PbrO2, however, sustained above the critical threshold of 10 mmHg. With intact autoregulation, CBF responds globally and remain above the ischemic threshold, which was confirmed by a physiological metabolic muster.

Controversially, in the group with moderate ICH volume (3.0ml), there was an elevated ICP directly after ICH induction resulting from the hemorrhage mass effect. This lead to reduced CBF in the PHZ locally. Subsequently CPP was affected with a global increase of ICP resulting in critical decreases of CBF. Therefore the PbrO2 was affected and decreased below the ischemic threshold. Furthermore, the ischemic pattern was correlated with pathological metabolic changes in PHZ including a significant increase in lactate and lactate/pyruvate ratio. These ischemic events occurred in the group with moderate ICH volume (3.0ml), but it did not occur in other groups. There were changes related to the ICH size. Further experiments with bigger ICH volume and longer observed time period may contribute more information about the above mentioned mechanisms.

Animal without ICH showed an elevated ICP after hemorrhage induction correlated with significant decrease of PbrO2, but in contrast to ICH, CBF as L/P ratio was not affected over a time period of 9 hours after hemorrhage induction. Hypothetically an acute hydrocephalus occurs leading to globally elevated ICP. Autoregulation mechanism may be still intact in this phase and therefore CPP and CBF respectively compensated for the globally elevated ICP.

Decompressive craniectomy can reduce an elevated ICP resulting from mass effect after ICH induction. We showed that CBF and metabolic events are not affected after ICH induction. In these open skull experiments we were able to detect signs of CSD. CSDs were seen as DC shifts, characteristics of neuronal depolarization with increasing energy consumption. As known from the literature, CSDs may have the potential to affect brain perfusion and increase the secondary injury.

Further experiments with mini craniotomy may contribute more information about the correlation between CSDs and other ischemic events. Translation into clinical research might be feasible in the near future.