Influence of sedation on cerebral uptake of glutamate following traumatic brain injury

Summary / Zusammenfassung

BACKGROUND:
Sedation and anesthesia significantly decrease neuronal release of transmitters as e.g., glutamate which reduces underlying brain damage. Apart from its function as an excitatory amino acid, glutamate also serves as an important metabolic protagonist within the intermediate metabolism improving energetic status.

MAIN HYPOTHESIS:
Prolonged sedation using different agents (midazolam or thiopental) differentially influences cerebral uptake and release of glutamate determined by calculated arterio- jugular venous differences.

STUDY DESIGN:
Daily analysis changes in arterial and jugular venous glutamate determined by HPLC in patients with severe traumatic brain injury.

Publications / Publikationen

Stover JF, Kempski OS.
Anesthesia increases circulating glutamate in neurosurgical patients.

Stover JF, Sakowitz OW, Kroppenstedt SN, Thomale UW, Kempski OS, Flügge G, Unterberg AW.
Differential effects of prolonged isoflurane anesthesia on plasma, extracellular, and CSF glutamate, neuronal activity, 125I-Mk801 NMDA receptor binding, and brain edema in traumatic brain-injured rats.

Stover JF, Kroppenstedt SN, Thomale UW, Kempski OS, Unterberg AW.
Isoflurane doubles plasma glutamate and increases posttraumatic brain edema.

Stover JF, Morganti-Kossmann MC, Lenzlinger PM, Stocker R, Kempski OS, Kossmann T.
Glutamate and taurine are increased in ventricular cerebrospinal fluid of severely brain-injured patients.

Stover JF, Pleines UE, Morganti-Kossmann MC, Stocker R, Kempski OS, Kossmann T.
Thiopental and midazolam do not seem to impede metabolism of glutamate in brain-injured patients.

Stover JF, Lowitzsch K, Kempski OS.
Cerebrospinal fluid hypoxanthine, xanthine and uric acid levels may reflect glutamate-mediated excitotoxicity in different neurological diseases.
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