Hypertonic sodium lactate

Hypertonic sodium lactate infusion (HL) in patients with TBI have shown HL can be successfully used to reduce secondary intracranial hypertension or as a valid alternative to mannitol for the treatment of elevated intracranial pressure (ICP).

Although HL may be a valid therapeutic intervention, the exact conditions and potential indications for its utilization are still to be better characterized. The cerebral microdialysis (CMD) lactate to pyruvate ratio (LPR) is a marker of tissue cerebral metabolic state. Elevated LPR >25 is a marker of impaired cerebral oxidative metabolism and outcome after TBI.

The CMD LPR would therefore be an ideal marker to identify subjects with high LPR who are more likely to benefit from therapies aiming to improve neuroenergetics, such as HL. On the other extent, the majority of animal and human data on HL is predominantly focused on TBI, and only a few tested models of cerebral ischemia [1].

Recent reports have advocated use of hypertonic sodium lactate, based on claims that it is glucose sparing and provides an oxidative fuel for injured brain. However, changes in extracellular concentrations in microdialysis are not evidence that a rise in extracellular glucose level is beneficial or that lactate is metabolized and improves neuroenergetics. The increase in glucose concentration may reflect inhibition of glycolysis, glycogenolysis, and pentose phosphate shunt pathway fluxes by lactate flooding in patients with mitochondrial dysfunction. In such cases, lactate will not be metabolizable and lactate flooding may be harmful. More rigorous approaches are required to evaluate metabolic and physiological effects of administration of hypertonic sodium lactate to brain-injured patients [2].

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2)
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