Cerebral metabolism

Changes in metabolism are known to contribute to tumor phenotypes. If and how metabolic alterations in brain tumours contribute to patient outcome is still poorly understood. Epigenetics impact metabolism and mitochondrial function.

Disturbed brain metabolism is a signature of primary damage and/or precipitates secondary injury processes after severe brain injury. Sedatives and analgesics target electrophysiological functioning and are as such well-known modulators of brain energy metabolism. Still unclear, however, is how sedatives impact glucose metabolism and whether they differentially influence brain metabolism in normally active, healthy brain and critically impaired, injured brain.

An intricate relationship normally exists between cerebral metabolism and energy substrate supply because of the brain's high substrate demand and limited storage capacity. In head-injured patients, this balance can be disrupted. The brain parenchyma directly involved by the injury is hypometabolic in respect to glucose and oxygen, whereas peri-injury tissue may have an elevated metabolic rate [1].

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Ritter AM, Robertson CS. Cerebral metabolism. Neurosurg Clin N Am. 1994 Oct;5(4):633-45. Review. PubMed PMID: 7827475.