Glucose

Glucose is conventionally regarded as the major energy substrate, although lactate can also be an energy source.

Blood glucose

see Blood glucose.

Glucose metabolism

see Glucose metabolism.

Glucose is an essential substrate for the brain and is also important for several pathways, which are crucial for brain cell survival, for example, the pentose-phosphate pathway.

Glucose crosses the blood brain barrier via specific glucose transporters (GLUT), proportionally to baseline metabolism and demand.

After TBI, energy demand is considerably augmented, causing an increase in cerebral metabolic rate of glucose and so-called hyperglycolysis.

This may ultimately lead to energy dysfunction or crisis, with concomitant critical depletion of cerebral extracellular glucose.

This phenomenon may be further aggravated when availability of systemic glucose is limited, such as by intensive insulin therapy.

Is a sugar with the molecular formula C6H12O6. The name “glucose” (/ˈgluːkoʊs/) comes from the Greek word γλευκος, meaning “sweet wine, must”.

The suffix “-ose” is a chemical classifier, denoting a carbohydrate. It is also known as dextrose or grape sugar. With 6 carbon atoms, it is classed as a hexose, a sub-category of monosaccharides. α-D-glucose is one of the 16 aldose stereoisomers. The D-isomer occurs widely in nature, but the L-isomer does not. Glucose is made during photosynthesis from water and carbon dioxide, using energy from sunlight. The reverse of the photosynthesis reaction, which releases this energy, is a very important source of power for cellular respiration. Glucose is stored as a polymer, in plants as starch and in animals as glycogen.

Although not a ketone itself, the concentration of beta hydroxybutyrate, like that of other ketone bodies, is raised in ketosis. The compound can be used as an energy source by the brain when blood glucose is low.

Cerebral energy dysfunction has recently emerged as an important determinant of prognosis following traumatic brain injury.

Although the exact mechanisms are still not completely understood, clinical investigation using the intra-cerebral microdialysis (CMD) technique has identified reduced cerebral extracellular glucose as a surrogate marker of post-TBI cerebral energy dysfunction and outcome.

The regional cerebral metabolic rate for glucose (rCMRglu) has never been investigated in large consecutive groups of patients with normal pressure hydrocephalus (NPH), a potentially treatable form of dementia with an unpredictable outcome after shunt surgery. Using PET and 18F-2-fluorodeoxyglucose, rCMRglu was studied in 18 patients who fulfilled hydrodynamic criteria for NPH and in whom a biopsy of the frontal cortex was obtained. When compared with an age matched group of 11 healthy subjects, the patients with NPH showed a significant rCMRglu reduction in all cortical and subcortical regions of interest. Individual metabolic patterns, however, disclosed a large topographical heterogeneity. Furthermore, histopathological examination identified Alzheimer's disease or cerebrovascular disease in six cases, and no parenchymal disease or non-specific degenerative processes in the remaining 12. After separating the patients according to the histological diagnosis, the rCMRglu patterns were still heterogeneous, the abnormalities ranging from focal to diffuse in both subgroups. After shunt operation, 11 patients did not improve or worsened clinically. Six patients improved; of those, two had Alzheimer changes and two cerebrovascular changes in their biopsy. The metabolic pattern of these six patients did not differ from the rest of the NPH group. The results indicate that the NPH syndrome may be non-specifically associated with different degenerative disorders. The metabolic heterogeneity, together with the heterogeneous histopathological findings, indicate the necessity of reevaluating the pathogenesis of the NPH syndrome, and may account for the high variability in the success rate of shunt surgery series [1].

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Tedeschi E, Hasselbalch SG, Waldemar G, Juhler M, Høgh P, Holm S, Garde L, Knudsen LL, Klinken L, Gjerris F, et al. Heterogeneous cerebral glucose metabolism in normal pressure hydrocephalus. J Neurol Neurosurg Psychiatry. 1995 Dec;59(6):608-15. PubMed PMID: 7500099; PubMed Central PMCID: PMC1073756.