Cerebral metabolic rate of oxygen

The brain is metabolically one of the most active of all organs in the body. This consumption of O2 provides the energy required for its intense physicochemical activity. The most reliable data on cerebral metabolic rate have been obtained in humans. Cerebral O2 consumption in normal, conscious, young men is approximately 3.5 ml/100 g brain/min.

The rate is similar in young women. The rate of O2 consumption by an entire brain of average weight (1,400 g) is then about 49 ml O2/min. The magnitude of this rate can be appreciated more fully when it is compared with the metabolic rate of the whole body. An average man weighs 70 kg and consumes about 250 ml O2/min in the basal state. Therefore, the brain, which represents only about 2% of total body weight, accounts for 20% of the resting total body O2 consumption. In children, the brain takes up an even larger fraction, as much as 50% in the middle of the first decade of life [1)].

The cerebral metabolic rate of oxygen consumption (CMRO2) arises from neurons utilizing energy for two functions:

1) maintenance of cell integrity (homeostasis) which normally accounts for ≈ 40% of energy consumption, and 2) conduction of electrical impulses. The occlusion of an artery produces a central core of ischemic tissue where the CMRO2 is not met. The oxygen deficiency precludes aerobic glycolysis and oxidative phosphorylation. ATP production declines and cell homeostasis cannot be maintained, and within minutes irreversible cell death occurs; a so-called cerebral infarction. Surrounding this central core is the penumbra, where collateral flow (usually through leptomeningeal vessels) provides marginal oxygenation which may impair cellular function without immediate irreversible damage. Cells in the penumbra may remain viable for hours.

The regional cerebral metabolic rate for glucose (rCMRglu) has never been investigated in large consecutive groups of patients with normal pressure hydrocephalus (NPH).

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 [2)].

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Many brain diseases have been linked to abnormal oxygen metabolism and blood perfusion; nevertheless, there is still a lack of robust diagnostic tools for directly imaging cerebral metabolic rate of oxygen (CMRO2) and cerebral blood flow (CBF), as well as the oxygen extraction fraction (OEF) that reflects the balance between CMRO(2) and CBF.

MR-derived cerebral metabolic rate of oxygen utilization (CMRO2) has been suggested to be analogous to PET-derived CMRO2 and therefore may be used for detection of viable tissue at risk for infarction.

MR-derived CMRO2 was decreased within diffusion-restricted tissue and stable within perfusion-impaired tissue, suggesting that this technique may be adequate to reveal different pathophysiological stages in acute stroke [3].

1)
http://www.ncbi.nlm.nih.gov/books/NBK28194/
2)
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3)
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