ALTERATIONS IN CEREBRAL AVAILABILITY OF METABOLIC SUBSTRATES AFTER HEAD TRAUMA. C.S. Robertson, J.C. Goodman, S. Bejot, CN Ou., Departments of Neurosurgery and Pathology, Baylor College of Medicine, Houston, Texas

The arterial whole blood concentration and the cerebral metabolic rate (CMR = arterial-jugular venous difference x cerebral blood flow) of oxygen, glucose, lactate, pyruvate, beta-hydroxybutyrate, and 16 amino acids were measured daily for the first 5 days after injury in 12 comatose head-injuried patients. Compared to normal fasting controls and to noncomatose patients undergoing elective craniotomy, head-injuried patients had increased arterial levels of glucose and lactate. Head-injuried patients had decreased levels of beta-hydroxybutyrate, isoleucine, leucine, valine, arginine, serine, and threonine. The hyperglycemia and lactic acidosis were most severe on day 1; the hypoaminoacidemia was most marked on day 2. CMRglucose was decreased to 60% of normal, and an increased proportion of glucose was anaerobically metabolized to lactate. On day 1 the normal net cerebral influx of amino acids was present. By day 2, when most of the amino acids had reached their nadir, there was a net efflux of amino acids from the brain. A significant correlation between the arterial concentration and the cerebral uptake of several metabolic substrates including beta-hydroxybutyrate, tyrosine, phenylalanine, isoleucine, leucine, and valine was demonstrated. The systemic response to head injury may influence which substrates are available for cerebral energy production and repair of injuries.

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