Hepatic Encephalopathy: Pathophysiology and Treatment (Experimental Biology and Medicine)

Roger F. Butterworth, Gilles Pomier Layrargues

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Hepatic Encephalopathy (HE) is a neuropsychiatric disorder resul t ing from liver failure. HE may be associated with fulminant (acute) hepatic failure or chronic liver disease with portal-systemic shunting. The latter condition is characterized neuropathologically by astro cytic rather than neuronal changes (Alzheimer Type II astrocytosis). The former is frequently accompanied by cerebral edema. Several hypotheses have been proposed to explain the pathogene sis of HE. These include: 1. A toxic action of a substance (or substances) such as ammonia on brain function 2. A deficit of cerebral energy metabolism 3. Neurotransmitter changes, and, more recently 4. The role of "endogenous benzodiazepines. " This volume summarizes the results of a symposium held in Val David, Quebec from October 3D-November I, 1988, that was devoted to an evaluation of the evidence for and against the various hypothe ses of HE. Data from studies in patients, in experimental (animal) models of HE, and in cultured cell preparations were discussed. In addition, a review of available approaches to the treatment and man agement of HE was included. The therapeutic use of lactulose, anti biotics, dietary treatment, and branched-chain amino acid treatments were included, as well as the results of preliminary studies of the therapeutic use of the benzodiazepine antagonist, flumazenil. Roger F. Butterworth, PhD Gilles Pomier Layrargues, MD v Acknowledgments The symposium was made possible by the generous financial assistance of: Hoffman-La Roche Ltd.

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