01.21.07

INTRAVENOUS PYRITINOL IN CLOSED HEAD INJURIES

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A double blind,prosprective clinical trial on the benefits and limitations of Pyritinol in head traumata subjected 50 cases (age 12-60) divided randomly into a group of control receiving conventional treatment such as steroids ,and one receiving Pyritinol additionally.
An intensive 10-day observation including regular checks on the Glasgow Coma Scale.motoric, cranial nerves,and higher functions status.the possiblity of increased intracranial pressure or hematomas,followed by a less intensive one for a period of 2-4 weeks,were recorded.
EEG,angiography and CT scans were performed if indicated,a.o. to exclude increased intracranial pressure and hematomas.
The results,in today's atmosphere of traumatic treatment favouring intensive care,will be discussed.

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01.21.08

DISTURBANCE OF BEHAVIOR AS RESIDUAL SYMPTOMATOLOGY AFTER SEVERE HEAD INJURY.

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Disturbance of personality and emotions in the remission phase after traumatic apallic syndrome often impede the reintegration of the patient in normal social life (BRAAKMANN, VanZOMEREN, Holland, BROOKS, BOND, Great Britain, JOCHHEIM, West Germany , NAJENSON, Israel). The reduction of emotional control accentuates premorbid personality traits. Often emotional disturbances are caused by lesions of the temporal lobes. The Klüver-Bucy syndrome as a severe form of post-traumatic behavorial disturbance is mostly reversible in the case of cortico-diencephalic disconnection. In the case of more severe structural lesions of a temporal lobe the disturbances of personality with disinhibition of emotional control, hypermetamorphosis, irritability and aggressive tendencies persist.
14 patients are reported, of whom 9 revealed extended lesions in a temporal lobe an 5 only mild lesions. Clinical findings and neuropsychologic test results are reported. A correlation between the morphological findings, clinical symptomatology and neuropsychologic test is made.

01.30.01

ETHANOL NEUROTOXICITY: EFFECTS ON EMBRYONIC NEURON PRODUCTION OF AND RESPONSE TO DIFFUSIBLE AND IMMOBILIZED NEUROTROPHIC MOLECULES

K. E. Dow

Prenatal ethanol exposure is associated with characteristic physical abnormalities and central nervous system dysfunction. The effects of ethanol on the developing nervous system occur during early neurogenesis as neurons interact with matrices of growth and with diffusible trophic factors within their milieu. An in vitro paradigm which quantitates neuron-matrix interaction, survival and process formation has been used to examine directly the effects of ethanol on embryonic neurons. Etnanol produced dose-dependent innibition of neuronal process formation on several biological substrates (poly-D-lysine, laminin, and neuron-produced substrate-attached materials or SAMs). This effect was seen at concentrations of ethanol lower than those previously shown to have toxic effects in vivo. Attachment of sensory and spinal cord neurons to the substrate and survival of substrate-attached neurons (measured by a colorimetric assay of mitochondrial activity) were not affected by ethanol on any of the substrates. Neuron-conditioned medium had lower specific activity of SAMs when produced in the presence of ethanol. The binding of Nerve Growth Factor (NGF) to its receptor on primary sensory neurons was not affected by ethanol.
These data suggest that ethanol may exert its toxicity during neurogenesis by altering the metabolic characteristics of neurons in such a way that, following attachment to appropriate substrates of growth, process formation and production of self-sustaining tropnic factors are inhibited.

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01.30.02

PREDOMINANTLY LIMBIC LOCALIZATION OF CORPORA AMYLACEA. QUANTITATIVE TOPOGRAPHY

M. Papp, I. Antalics, L. Kutor and O. Major

Alder (1955) J Ment Sci 99, 0897 noticed that corpora amylacea (CA) were concentrated in structures in proximity to the CSF, particularly in the hippocampus. Our routine histopathological examination of about 100 human brains gave the impression of CA accumulation within limbic structures rather than in the somatic brain. Therefore, eight of them with various neurological diseases were used for quantitative estimation of CA. Counting of CA was made in every 0.2 mm^2 of 5 micron thick coronal sections including both hemispheres using a light microscope equipped with a specially designated calculating device. Although the number of CA varied from brain to brain, the basic pattern of their quantitative topography was similar. Accordingly, 75-95 % of CA were concentrated in certain limbic structures identified according to McLean, Papez and Livingston (1952 EEG Clin Neurophysiol 4, 407, 1958 Reticular Formation Brain Stem 591 pp, 1971 Arch Neurol 24, 17). Subependymal limbic structures constantly accumulating CA were the septum, stria terminalis and the fornix. Those facing to the subarachnoid cisterns were the olfactory trigone, anterior perforated substance, hippocampus, orbito-frontal, dorso-temporal and anterior insular cortex. Contrary to this, the somatic brain, even in the proximity of CSF, e.g. the neocortex contained only 20 % or so of the sum of CA.

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