MS-C4

MS-C4-03

The process of brain swelling or cerebral edema is the most common reaction to head injury and is seen as a constant accompaniment to each other form of brain injury. Immediately after trauma, the blood brain barrier breaks down at the site of the injury. Generally, local vasculature is disrupted and blood cells and serum proteins massively invade the injuried area. About 24hr postlesion CNS edema is patent. If edema is not compensable its mass and pressure effects may affect vascular perfusion, resulting in secondary ischemia of the injured region which will increase the edema. The traumatic edema may spread outside the traumatized region and involve the whole hemisphere or the whole brain. In its instance there is a grave risk of excessive and irreversible increase in intracranial pressure, herniation and perfusion failure which may lead to brain death.In the child cerebral edema occur in connection with even apparently mild injury, and may lead to death.

The resolution of posttraumatic edema is triggered by different mechanisms. For proteins the macrophagic activity of astrocytes seems to play a role. A bulk flow along the perivasculary and extracellulary space towards the ventricle and the subarachnoidal space. In adults, cerebral edema of a traumatic origin, if long standing, may have deleterious effects on the white matter and lead to demyelination. Histologically, depending of the duration and severity of the edema, the microscopic appearance of an edematous area is usually spongy with numerous vacuoles in the tissue Because most edema fluid is rather low in protein content, the edematous tissue usually looks paler than normal white matter in H&E preparations. Long-standing edema, of more than several weeks, may lead to reactive gliosis in the affected region.

MS-C4-04

CLINICAL BIOCHEMISTRY OF BRAIN INJURY

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Abstract not submitted

MS-C4-05

New approaches in the diagnosis of acute and subacute head-injury

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Depending on the direction of the impact of force in head-injuries - which could be multiple, if the head is knocked more than once - as well as on the intensity of the impact of force, a different traumatic pattern may be the result: outer linear cerebral trauma (Grcevic), lower inner cerebral trauma (Lindenberg) and rotational cerebral trauma (Pudenz-Sholden). The developement of an apallic syndrome may be the result of an acute damage of the hemispheres and the brainstem. An acute midbrain-syndrome (Gerstenbrand, Lücking) is an obligate prodrome of the the apallic syndrome. It is to say that the five stages very often occur in an uniform course, in rarer cases a bulbar brain syndrome until stage II may have existed.
A prolonged midbrain syndrome is possible as a partial apallic syndrome.The remission after an apallic syndrome shows eight stages according to the Innsbruck Remission Scale. After having reached the third remission-phase the prognosis mostly is a good one, but a defective state of different levels is possible and even a complete remission might be the result.
For the course of the remission it is essential to minimize the secondary noxa,e.g. brainedema, hypoxie etc., to prevent tertiary noxa, e.g. malnutrition, bedrest syndrome etc., and to discover as early as possible quarterly noxa, e.g. occluded hydrocephalis, hygrama, brain abscess etc.

MS-C4-06

The value of neurophysiological tests in the course of acute and subacute brain injury.

Different neurophysiological tests are used in posttraumatic coma. Some of them are influenced by sedative drugs, especially the EEG, which only can be used for monitoring barbiturate coma, despite its high prognostic value in unsedated patients. Also the blinkreflex is severely impaired by sedation. Especially, the R_2 components are suppressed such indicating a "false" bad outcome.

Little or no influence is seen in BAEP and SSEP studies. The prognostic value is high. Measurement of interwave latencies excludes affection by limb temperature, peripheral neuropathies or damage to the ear and ensures adequate stimulation. Less artifacts are found in sedated patients, but the methods can also be used in subacute brain injury, when sedation ist stopped. MEPs do not add further information of prognostic significance.

The choice of the method used for determening brain function largely depends on the kind of therapy, but also on the time course of coma. In acute brain injury SSEPs and BAEPs are prefered, while in subacute stages all methods are of considerable prognostic value.

Second Congress of the Paneuropean Society of Neurology, Vienna, december 7-12, 1991 : book of abstracts

Autor: Franz Fazekas; Erich Schmutzhard; Karl Zeiler

Verlag: Hofburg : Vienna ; [s.n.], 1991.

Ausgabe/Medienart: Gedrucktes Buch : Tagungsband : Englisch

Bewertung: (noch nicht bewertet)

0 mit Rezensionen - Verfassen Sie als [redacted]

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Details

Medienart:	Tagungsband
Dokumenttyp	Buch
Alle Autoren:	Franz Fazekas; Erich Schmutzhard; Karl Zeiler
	Weitere Informationen zu: Franz Fazekas Suchen
OCLC-Nummer:	803982114
Anmerkungen:	Na okł. : A congress in the decade of the brain.
Beschreibung:	239, [1] ; il. ; 30 cm.
Andere Titel	Congress of the Paneuropean Society of Neurology, 2
Verfasserangabe:	eds F. Fazekas, E. Schmutzhard, K. Zeiler.