New trends in clinical neuropharmacology, ed. D. Bartko *et al.* © John Libbey & Co Ltd, 1988, pp. 71-77.

THE MANAGEMENT OF HEAD INJURY

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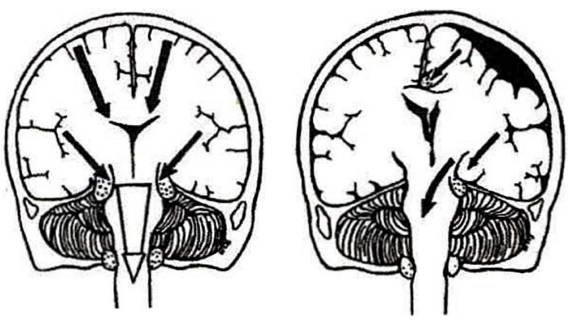
More than half a million craniocervical injuries were recorded in 1986 in the states of the common market. Over 50 % of them were caused by traffic accidents and about 25 % by accidents at work. Increasing severity of the trauma indicates a higher probability of concomitant head injury, which will become, in more than 50 % of cases, the main injury. Patients after head injury, with decreased consciousness or coma, show different stages of a traumatic midbrain syndrome or bulbar-brain syndrome. Worsening of the clinical feature is connected with the involvement of lower brainstem structures due to the increasing intracranial pressure (ICP) caused by a primary lesion itself, or, as a consequence, by the secondary lesion (Tab. 1,2).

Primary lesions after brain injury	secondary lesions after brain injury
-cortical contusion	-ischemia by shock
-brain laceration	-perifocal/local brain edema
-intracerebral/extracerebral bleeding (ICH, SDH, EDH, SAH)	-->local/diffuse brain atrophy
-periventricular lesions	-->tentorial/foraminal herniation
-rhexis of corpus callosum	--->local midbrain lesion
-brainstem contusion	--->ischemic local lesions by strangled arteries
	--->haemorrhage due to passive hyperemia

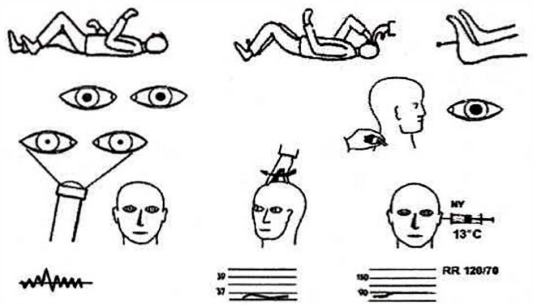
Tab. 1 & 2: Primary and secondary lesions after brain injury.

The clinical feature of the acute midbrain syndrome shows two forms of development due to the pathomechanism of the herniation (Fig. 1 below).



PATHOMECHANISM OF..
--> uncal herniation
--> central (medial) herniation

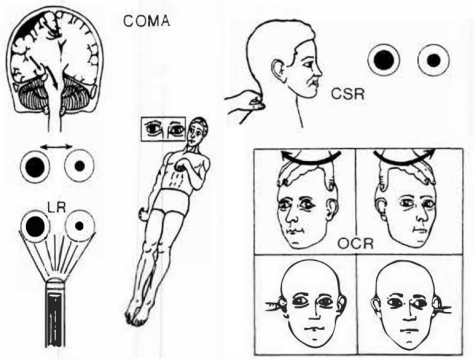
Both, the uncal and the central herniation lead to a characteristic sequence of symptoms, which are dependant on the level of the lesion at the brain stem.
The medial (central) herniation: Increasing general pressura induces pressure of the medial parts of the temporal lobe on to the upper brain stem. This induces a downward displacement of the brain stem structures. This transtentorial herniation leads to local direct pressure of the brain stem structures, strangulation of the arteries with ischamia and haemorrhages due to passive hyperemia. Due to this progressive lesioning of brain stem structures, a disengagement of brain stem functions of cortical control is effected. Therefore cortical uncontrolled mesencephalic primitive motor patterns, disturbances of vegetative functions, optomotoric and of the ascending reticular system with typical postures, disturbed eye movements and position, vegetative symptoms and consciousness occur.



COMA
NO REACTION TO ACOUSTIC STIMULUS
FLEXION UPPER/EXTENSION LOWER LIMBS
REDUCED PUPIL SEIZE
MUSCLE TONE INCREASED
BEGINNING DECOMPENSATION OF THE VEGETATIVUM

Fig. 2: Typical symptoms of MBS 3 (central herniation)

The uncal herniation: The consequence of a local pressure on the brain is the uncal herniation, also with characteristic features. It is described as the first and second stage of lateralisation of acute midbrain syndrome - LMBS 1 & 2.
These lateralized midbrain syndrome phases show a typical homolateral lesion of the nervus oculomotorius and a contralateral lesion of the midbrain structures, which are pressed to the edge of the tentorium. The second phase of LMBS is followed by a medial midbrain syndrome stage 4, but with remaining pupillar difference as a residual marker of lateralisation (Fig. 3).
Insistent increased ICP causes herniation into the foramen magnum, with lesion and disintegration of the bulbar brain and the clinical feature of bulbar brain syndrome (Fig. 4).



COMA

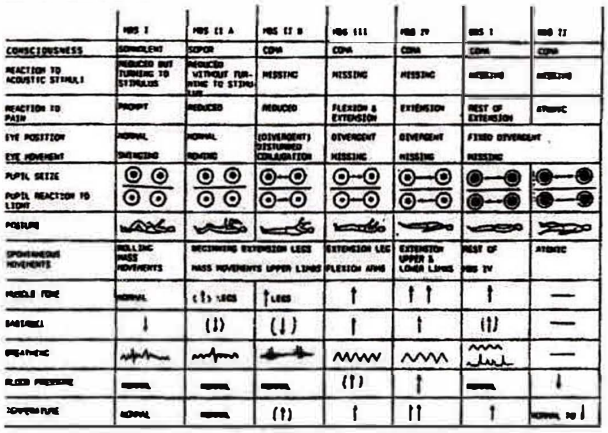
HOMOLATERAL WIDE PUPIL WITH

NO REACTION TO LIGHT

EXTENSION UPPER & LOWER LIMBS HOMOLATERAL

FLEXION UPPER AND EXTENSION LOWER LIMBS

KONTRALATERAL



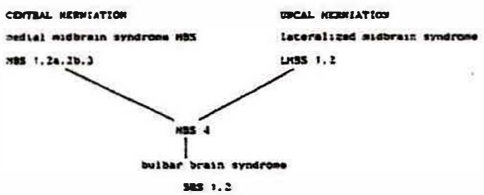
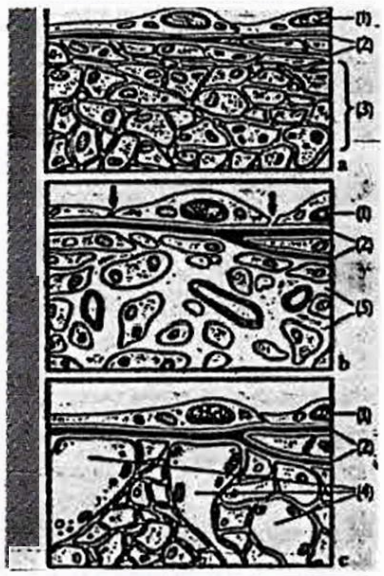


Fig.: 3,3a,4
symptoms of lateralized midbrain syndrome 2
symptomatology of MBS 1 to BBS 2
development of central and uncal herniation

The first step of the therapy of severe head injury with the symptomatology of a midbrain syndrome should be the reanimation phase with coma monitoring, stabilisation of organ function and the differentiation between primary (intracranial hemorrhage) and secondary lesions (local or diffuse edema). One of the most important factors for a successful treatment but also for the first steps of rehabilitation is the initiation of therapy at the earliest staga of midbrain syndrome. Therefore the diagnose of the cause of the herniation then the clinical feature with coma monitoring becomes the time decisive factor. The progressive slipping off to lower stages of midbrain has to be stopped as early as possible. In our experiences diffuse brain edema seems to be the most

dangerous factor after brain trauma, with a frightening by fast slipping off into midbrain syndrome stage 4 or bulbar brain syndrome, especially in young patients.



FORMS OF
CEREBRAL EDEMA

1. vasogenic edema
   affects mostly the white matter
   the form of edema after brain
   injury
2. cytotoxic edema
   ICV increase of neurons and
   glia in the gray matter
3. interstitial edema
   diffusion of a vasogenic edema
   from a local lesion into regions
   with intact barriers

Fig. 5: Forms of cerebral edema.

For the reduction of ICP a decrease of the volume of each intracranial compartement - spinal fluid, extra- and intracellular volume - is aimed for. For the therapy of brain edema the use of general therapeutic measures such as sufficient ventilation, special head-trunk position and balanced arterial pressure, beside that hyperventilation, osmotic agents, barbidiuretics and steroids proved to be sufficient (Tab. 3,.Tab. 4).

The use of osmotic agents is bound to intact barrier between extra- and intracellular volume (blood-brain barrier). The disadvantage of the therapy with osmotic agents and especially with polyvalent alcohol substances is a rebound phenomenon, which is caused by metabolism of the substance in the intracellular volume. The consequence is a disarangement of the solvent drag into the cell. Renal problems will occur when reaching a osmolarity above 320 mosmol/kg. As a side effect of sorbit (specially in combination with hyperventilation) lactat acidosis can be seen in some cases (Tab. 5, Tab. 6).

Barbiturates, possibly, have an effect on the metabolism of brain cells and also a direct effect on the cerebral vessels. EEG control (burst suppression) seems to be the best technique for ascertaining the required loading dose of the barbiturate, but a randomized clinical study is not known by the authors (Tab. 7).

THERAPY OF BRAIN EDEMA

1.GENERAL THERAPEUTIC MEASURES
2.HYPERVENTILATION
3.OSMOTIC AGENTS
4.BARBITURATES
5.DIURETICS
6.STEROIDS

HYPERVENTILATION

$PaCO_2$ 25-30mmHg
PaO_2 75-80mmHg
CONTRACTION OF CEREBRAL VESSELS
INCREASING BLOOD SUPPLY
IN THE LESIONED AREA

GENERAL THERAPEUTIC MEASURES

1.SUFFICIENT VENTILATION
$PaCO_2$ not > 42mmHg
PaO_2 not < 80mmHg
2.HEAD / TRUNK POSITION
ELEVATED AT 30°
3.AVOIDANCE OF INCREASE OR
DECREASE OF ARTERIAL PRESSURE

OSMOTIC AGENTS

MANNITOL 20%
50-100ml in about 15min iv./5h
SORBITOL 40%
0.5-2 g/kg iv in about 30min
CAVE: lactat acidosis
GLYCERIN 10%
0.2 g/kg/h iv
nearly without side effects
GLUCOSE 50% (obsolet)
FRUCTOSE 40% (obsolet)
LAEVULOSE 40% (obsolet)
UREA
5-16 mg/min iv
CAVE:problems with coagulation
haemoglobinuria

DIURETICS

FUROSEMID
0.25-0.30 mg /kg iv.
ACETAZOLAMIDE
5-20 mg /kg iv
not usefull as monotherapy

STEROIDS

dexamethason
100 mg loading dose
4-8 mg/ 6h continuing

BARBITURATES

pentobarb.cone
3-5 mg /kg loading dose
1 mg /kg continued
thiopentone
15-20 mg / kg loading dose
5 mg /kg/h for the next 6h
2-3 mg /kg/h following

Tab. 3,4,5,6,7 therapy of the acute midbrain syndrome

An acute midbrain syndrome or lateralized midbrain syndrome (see also Fig.4) can desintegrate to bulbarbrain syndrome or brain death, but also develop to a prolonged midbrain syndrome (specially from MBS 2b, 3 or 4) or to an apallic syndrome with 3 intermediate stages (coma prolonge,parasomnia,akinetic mutism). A reintegration is possible from a deep stage of traumatic midbrain syndrome with remittance of symptoms.

More often a prolonged midbrain syndrome or a apallic syndrome develops. The prolonged midbrain syndrome but also the

apallic syndrome show seven stages of remission, ending in a"defect stage" with remaining symptoms or a restitutio (in 17% of cases with apallic syndrome) Fig.: 5. The development of the apallic syndrome is characterized by typical clinical symptoms, Tab.: 8 , but also by the onset of a overactivity of the sympathic nervous system. The clinical problems, which occur now we subsummarize as tertiary and quartary lesions after brain injury Tab.: 9,10. The first signs of a remission should be seen within the first 4-5 month after the trauma, in default whereof the prognose is bad.
Without special treatment the sympathic catabolic situation leads to marasm and severe peripheral and central nervous system lesions. Even high caloric nutrition can not stop this. The plasma level of norepinephrine is extremly high in these patients. Treatment with beta blocking agents and debrisoquine supresses the catabolic drive and allows a normocaloric nutrition. The basic principles of the programme for rehabilitation, which almost should beginn at the time of the intensive care, we show in Tab.: 11

SYMPTOMS OF THE APALLIC SYNDROME
coma vigile
missing emotional reaction
primitive motor pattern
fixed posture
increased sympathotonus
regulated rythm of phases of sleep and wakeness

Tab.:8 clinical symptoms of an apallic syndrome

TERTIARY LESIONS AFTER BRAIN INJURY	QUARTARY LESIONS AFTER BRAIN INJURY
origin: malnutrition	hydrocephalus aresorptivus
malabsorption	hygroma
electrolyte dysbalance	abscess
hypovitaminosis	meningoencephalistis per continuitatem or embolic
neuroendocrine dysfunction	KOMPLIKATIONS
superinfection	decubitus
	contractures
-->encephalopathy	periarticular ossifications
-->pontine myelinolyse	
-->myelopathy	
-->neuropathy	

Tab.: 9,10 tertiary and quartary lesions after brain injury

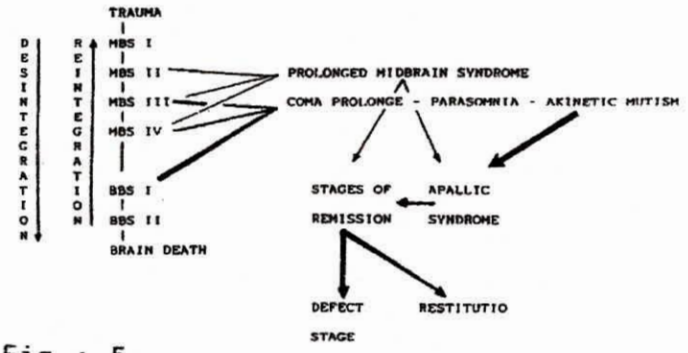


Fig.: 5

PROGRAMME FOR REHABILITATION

I.CARE

EARLY DIAGNOSE AND PREVENTION

OF QUARTARY LESIONS

II.NUTRITION

USING BETA - BLOCKERS 2500-3000kcal/d

WITHOUT 5000-7000kcal

(toxic complications)

III.PHARMACOLOGICAL TREATMENT

BETA BLOCKERS

HGH (only to use after Arginin

stimulation test)

DOPA-SUBSTITUTION

ANTISPASTICS

ANTIEPILEPTICS

BRAIN METABOLISM

ACTIVATING SUBSTANCES AND

VITAMIN B

IV.PHYSICAL THERAPY

VI.ERGOTHERAPY

VIII.RESOCIALISATION

V.STIMULATING METHODS

VII.LOGOPEDIC TREATMENT

IX.PSYCHOLOGICAL

SUPPORTMENT

HGH

HGH LEVELS ARE LOW IN ALL PATIENTS WITHOUT SPECIFIC STIMULATION

AFTER ARGININ STIMULATION HGH RELEASE INCREASES IN PATIENTS

WITH MBS II AND III, BUT NOT IN PATIENTS WITH MBS IV OR APALLIC

SYNDROME

TREATMENT OF SPASTICITY

TIZANIDINE 6-12 mg /d

BACLOPHEN 45-75 mg /d

BENZODIAZEPINE

DANTROLENE

Tab.: 11

NEW TRENDS IN CLINICAL NEUROPHARMACOLOGY

Calcium antagonists, acute neurology, headache and movement disorders

Proceedings of the Vth European Workshop on Clinical Neuropharmacology, Bratislava, July 6–8, 1987

Edited by

DANIEL BARTKO
PETER TURČÁNI
GERALD STERN

John Libbey
LONDON · PARIS

British Library Cataloguing in Publication Data
European Workshop on Clinical Neuropharmacology
(5th: 1987: Bratislava, Czechoslovakia)
New trends in clinical neuropharmacology
1. Man. Nervous system. Drug Therapy
I. Title II. Bartko, Daniel III. Turčáni, Peter
IV. Stern, Gerald V. Series
616.8'0461

ISBN 0-86196-146-3

First published in 1988 by
John Libbey & Company Ltd
80/84 Bondway, London SW8 1SF, England. (01) 582 5266
John Libbey Eurotext Ltd
6 rue Blanche, 92129 Montrouge, France. (1) 47 35 85 52

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Typesetting by E. E. Owens & Co Ltd.
Printed in Great Britain by
Whitstable Litho Printers Ltd, Whitstable, Kent.