Severe brain injury and intensive-therapeutic measures

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Epidemiology:

Brain injuries are a frequent cause of morbidity and mortality in European countries. The incidence of traumatic brain injuries **(TBI)** varies between 229 and 1.967/100.000 inhabitants. Similar numbers were reported for Russia. The highest incidence is reported in males between 15 and 24 years of age. TBI is the most frequent cause of death in the age group under 45 years.

We must differentiate between a closed cerebral trauma and an open brain injury caused by a penetrating object (stab, axe injury, injury by a bullet, etc.). Closed cerebral trauma can lead to a fracture of the scull if the impact is strong enough.

Biomechanics of traumatic brain injury:

The biomechanical basis of an impact on the skull was studied by SELLIER and UNTERHARNSCHEIDT in 1963 using a model of an iron ball filled with paraffin. Two physical factors play a role, the speed v and the acceleration b ($b = v^2 / 2 s$); s is the deceleration distance. An impact on the head causes a change of pressure inside the skull. The pressure is positive at the impact pole and negative at the counter pole, in between there is a zero-pressure zone.
At the impact pole and at the counter pole, damages emerge on the brain surface, called contusion zones. At the counter pole, negative pressure causes tissue damage due to gas bubbles; gas is solved in tissue under normal pressure. At the counter pole, direct damage due to contact of the skull bone leads to lesions on the surface of the brain. At the moment when the force acts on the head, the skull bone undergoes an elastic deformation in the impact region. When the deformed skull bone snaps back into its original position, damages of superficial cortical layers emerge following negative pressure due to gas bubbles. The damaged tissue of the brain usually reaches only a few millimeters into the depth, depending on the intensity of the acting force. The head, which is mobile on the cervical spine, carries out a rotational movement after an impact with a counter movement of the brain inside the skull, causing "shear forces" in the brain and at the brain vessels.

Depending on the direction of the impact, one can distinguish between *"linear brain* injury"(GRCEVIC) and a *"rotational* trauma"(PUDENZ-SHELDON. In most cases, a combination of these two traumatic patterns can be found.

According to the nature of the impact, an *"acceleration trauma"* and "*deceleration trauma' has to* be differentiated. In acceleration trauma the impact hits the partly mobile head in a static position, whereas in a deceleration trauma the head is stopped suddenly.

Of great importance for the analysis of the forces acting on the brain is the documentation of the impact and its location, and whether it was a single or a repeated impact. The scheme introduced by SPATZ in 1936, in the form modified in Innsbruck, has been useful up to now. We differentiate 6 types of an impact as follows. In type I the impact hits occipitally, in type II frontally including the face, in types II and IV from the side (left and/or right), in type V from above and in type VI from below.

Based on neuropathological findings in combination with the use of MRI examination, Grcevic and his group suggested a classification into linear outer brain trauma with cortical and subcortical tissue damages and linear inner brain trauma with damages in the interior of the brain. The linear outer brain trauma causes lesions in the coup and contre-coup region mostly destroying more tissue in the contre-coup region.

The linear inner brain trauma has to be divided into the linear inner upper brain trauma (type II, II a) with lesions of a "butterfly" type around the ventricles and the linear inner lower brain trauma (type V, Va) with lesions in the upper brain stem and the surrounding region (medial temporal lobe, diencephalon, and cerebellum). The linear inner lower brain trauma causes severest symptoms mostly with fatal and clinical course and the development of a traumatic apallic syndrome in many of these cases.

A Rotational trauma (PUDENZ-SHELDON) causes ruptures inside the brain (lacerations), intra- or extracerebral hematomas (acute and chronic hematoma, acute epidural hematoma) with consequential supratentorial mass movement.

Different forms of lesions after traumatic brain injury:

The primary traumatic brain lesion occurs during the moment of the impact to the head or with the penetration in case of open brain injury. These primary traumatic damages are irreversible and are called umbra, epicenter.

Around the primary brain damages there is a zone of partly damaged brain tissue decreasing towards the periphery (penumbra or periepicenter). The penumbra is the battlefield for therapy in the acute phase. In part of the penumbra secondary lesions develop. The so called secondary lesions are also diffuse tissue damages following hypoxia, hypoxemia and brain edema. Localized secondary lesions are found in the irrigation area of occluded cerebral arteries and blocked cerebral veins as well as around intracerebral or extracerebral hematoma. Secondary traumatic brain damage is partly treatable, if the treatment is initiated as soon as possible (oxygen supply, stabilizing of blood circulation, diminishing of brain metabolism).

Tertiary brain lesions develop in the post-acute and in the remission phase, mostly in severest TBI caused by malnutrition, malabsorption, avitaminosis, bed rest syndrome and as the critical .care neuropathy. Tertiary lesions include encephalopathy, pontine myelinolyse, myelopathy and

mostly polyneuropathy. Tertiary lesions are generally of "iatrogenic" origin and may totally diminish in the remission process of severe and severest brain injury.

As quaternary lesions hydrocephalus occlusus, traumatic meningoencephalitis and brain abscess are to be summarized. They can be controlled by treatment, if \diagnosed early enough.

Complications after TBI are joint contraction, periarticular ossification, pressure lesions of single peripheral nerves and decubitus. Complications are treatable, decubitus is not acceptable nowadays.

Classification of brain injury:

Modern classification has changed the earlier terminology like "brain commotion" (commotio cerebri), "brain contusion" (contusio cerebri) and "brain compression" to a four-grade system, "mild, moderate, severe and severest TBI". Following the trend to use a coma scale, mostly the Glasgow Coma Scale (GCS) with 15 points, mild traumatic brain injury (MTBI) has 13-15 points, moderate TBI 9-12, severe TBI 57. The Glasgow Coma Scale does not differentiate severest TBI with brain stem symptoms; the score must be under 5. The Neurological University Clinic Innsbruck developed another scale for TBI, the so called Innsbruck Coma Scale allowing a highly accurate prediction of non-survival in patients with scores of 0 or 1 even at the time of first examination after trauma.

Principally, patients with severest TBI are treatable. Most of them develop an apallic syndrome; direct remission after an acute brain stem syndrome is seldom. Patients are in danger of developing an irreversible breakdown of all brain functions (brain death). The GCS in its recent form is not reliable for a clear prognosis.
In moderate, severe and severest TBI, an immediate, well-prepared therapy is necessary. All these patients have to be brought to a neurorehabilitation center and should be treated according to an individual program. The acute therapy of severest brain injury patients requires treatment in an intensive care unit (ICU).

Severest brain injury, with primary or secondary traumatic damage of the brain stem:

Primary traumatic damages of the upper brain stem are the consequence of an impact type V and Va with the pattern of a linear inner lower brain trauma (LINDENBERG).
Secondary upper brain stem lesions are caused by tentorial herniation after supratentorial volume increase due to extra-, or intracerebral hematoma and/or diffuse or multilocular brain edema. The symptoms of primary and secondary upper brain stem lesions are uniform, showing the acute midbrain syndrome (midbrain-upper pons stage according to PLUM and POSNER); due to continuing volume increase foraminal herniation occurs, with symptoms of acute bulbar brain syndrome (the medullary stage according PLUM and POSNER.
In central tentorial herniation, the increased supratentorial pressure causes a displacement of both medial parts of the temporal lobe in the tentorium, followed by pressure on the upper brain stem). Simultaneously, a downward displacement of the total brain stem with traction of the

oculomotor nerve and the perforating brain vessels develops. In case of a one-sided supratentorial volume increase a cingular herniation occurs with shifting of the brain under the falx, followed by shifting of homolateral medial parts of the temporal lobe through the tentorium, with local pressure on the upper brain stem against the sharp edge of the opposite tentorium as well as on and the supplying vessels and the oculomotor nerve. In foraminal herniation, a displacement of the cerebellar tonsils into the foramen occipitale magnum emerges.

The symptoms of the **acute midbrain syndrome** develop in 5 phases. Mostly only two phases can be observed in case of primary lesion of the upper brain stem (phase III and IV). In the lateralized acute midbrain syndrome two phases have to be differentiated with transfer into the full stage of an acute midbrain syndrome. In acute bulbar brain syndrome two phases have to be observed.

During the development of the upper brain stem damage due to the tentorial herniation, continuing pressure on the midbrain or after the primary local lesion at the upper brain stem, a disinhibition of the motor and vegetative centers in the upper brain stem followed by the bulbar brain occurs, combined with dysfunction of the reticular formation succeeded by a continuous disturbance of consciousness up to coma

During the five phases of the acute midbrain syndrome, somnolence up to coma develops together with optomotor disturbances (eye position, pupils, vestibulo-oculomotor reflexes). The motor disturbances of extremities and trunk begin with mass movements, developing a flexed-stretch position of the extremities, progressing to a stretch position with stretch synergisms in the extremities and the trunk. The vegetative dysregulation manifests itself in an increase of respiration, blood pressure, pulse and body temperature with machine-like respiration. In the full stage, a pulse rate of more than 150, increased temperature up to 39° and massive increase of blood pressure can be observed.

In the **acute bulbar brain syndrome** (phases **I** and **II),** the motor and vegetative regulation centers fail to work. A continuation of coma with fixed divergent bulbi, highly extended pupils without reaction to light, flaccid posture of extremities with areflexia and - as most important symptom - respiratory arrest occur together with dropping of blood pressure and temperature. In rare cases the symptoms of an acute bulbar brain syndrome a direct remission is possible with or less severe defects. If the bulbar brain syndrome in its full stage continues for more than two hours usually an irreversible breakdown of the brain and brain stem functions, the brain death syndrome develops. This has to be confirmed by a zero-line in the EEG for a certain recording time and loss of the cerebral blood circulation needs to be demonstrated by TCD.

A **one-sided tentorial herniation,** uncal herniation produces a lateralization of an acute midbrain syndrome (third nerve stage — after PLUM and POSNER) in two phases. The first phase is characterized by deviation of the head and the bulbs to the opposite side of one-side-extended pupil and a flexed-stretching position of the extremities homolateral, with mass

movements contralateral, in the second phase stretch positions of the extremities homolateral with fixed stretch position on the contralateral side and extended pupils. Reduced consciousness up to coma evolves, in the further course the full stage of a midbrain syndrome develops.

Management of severest traumatic brain injury:

The primary task in an unconsciousness patient is to maintain and secure the vital functions. Orotracheal intubation should be used generously. In order to evaluate the condition of the patient, the Glasgow Coma Scale along with the clinical neurological examination is of a certain support as well as the Innsbruck Coma Scale.

The primary traumatic brain lesions cannot be influenced by any therapeutic activity. The brain tissue is irreversibly damaged. The intensity of brain tissue destruction and the accompanying brain edema influence the outcome of the patient. The aim of every activity in the periacute phase is to keep the patient alive and to diminish secondary brain lesions. Intensive oxygenation and sufficiently functioning blood circulation is the primary obligation. Cerebral ischemia due to shock or breakdown of the autoregulation in cerebral blood flow leads to secondary brain lesions. First aid on the site of the accident has to take this into consideration with all necessary actions for oxygen support, mainly to overcome the respiratory disturbances. The second imperative action is to preserve or diminish the brain edema.

The management of severest **TBI** has to follow ***four phases,*** the preclinical management, immediate measures in the admitting hospital, first intensive care measures and monitoring in the intensive care unit (ICU).

The preclinical management, acute measures:

At the site of the accident respiration hypercapnia, hypocapnia, obstructions in the respiratory tract etc., state of consciousness (somnolence, coma), global neurological status (pupils, motor behavior, stretch cramps, reactions to pain stimuli), documentation of the impact on the head (SPATZ -schema) have to be registered. Main actions to preserve free breathing (intubation if necessary), stabilisation of blood circulation (infusion, medicaments, etc.) have to be taken. If symptoms of midbrain syndrome are observed, an analgesic sedation should be initiated. The preclinical management is the responsibility of the first aid team.

Immediate measures in the admitting hospital:

The main program is control of ventilation, central venous catheter, bladder catheterization and stomach catheter. A detailed neurological status has to be performed by a neurologist (check list). Additional examinations are cerebral CT, X-ray of cervical spine, skull, if necessary of the extremities. Most important is the decision whether the patient should be transferred to an ICU as an urgent case. This decision must be taken, if there are symptoms of an acute midbrain or acute bulbar brain syndrome and GCS <8. The initiated treatment program has to be continued.

Intensive care measures:
For all patients with severest TBI GCS <8 a transfer to an ICU is mandatory. Besides monitoring and control of the vital functions, clinical monitoring of the neurological status using checklists is the central obligation. EEG recording is an additional support; evoked potentials can inform about brainstem and spinal cord lesions. Neurosurgical control has to be initiated as soon as possible. All laboratory tests have to be performed (permanent control of blood oxygenation). In cases with symptoms of a primary or secondary brain stem syndrome, monitoring of the intracranial pressure (ICP) must be started. Different ways of ICP (epidural, subdural, parenchymal, intraventricular) are possible. Intraventricular pressure measurement should be preferred, with the possibility to examine the CSF, but needs special technical know-how. The patient's treatment program has to be continued, analgesic sedation and brain edema therapy are the main points.

Monitoring and treatment in the ICU:
The main tasks are maintaining sufficient oxygenation and lowering brain pressure, brain edema therapy; in the therapeutic program the continuation of analgesic sedation is central. It is preferable to use high doses of barbiturates leading to barbiturate coma, continuing the basic idea to lower brain metabolism and to support the regeneration of the damaged neuronal systems. Artificial respiration may be necessary (pa CO) > 80). Extubating after 4-6 days, a tracheostomy and substitution of blood circulation (volume substitution, medicaments) should be applied.

A most important program is to find the adequate nutrition. Because of increased basic metabolism hypercalorization is necessary with a daily rate of 3.000 to 4.000 calories; if possible beta-blockers should be used.

As general support in the intensive care management, a special program is necessary for the body posture. It is recommended to bring the patient in a raised position of the upper trunk. The change of posture needs a fixed daily program. These methods are important for the prevention of the bed rest syndrome.

Early rehabilitation:
This has to start during the first days in the ICU. Methods performed are passive movements of the extremities, change of body posture, etc. Foot sole stimulation by pressure or vibration devices (stimulation shoe) have proved successful. Electrostimulation of the muscles of the extremities to prevent atrophy is being discussed.

Brain edema therapy:
This is a central point in the therapy. For osmotherapy, Mannit (20 %), Sorbit and Glyceresteril have proved to lower the intracranial pressure. Application of a bolus of Mannitol can be effective. Corticosteroids in high doses during the early phases can be successful. Hyperventilation is rejected as preventive method, brief hyperventilation therapy is

recommended. If the brain pressure cannot be controlled, mild hypothermia (33-35°C) has been increasingly discussed lately. If it is impossible to lower the intracranial pressure, a neurosurgical intervention, bilateral craniotomy can be performed.

Post-intensive care:

For the decision to dismiss a patient with a severest brain injury from the ICU, a control program for evaluation of the neurological and general deficit has to be started in close cooperation with the whole team. As soon as possible the patient has to be transferred to a neurological ward with experience in rehabilitation of brain injured patients. Patients with an apallic syndrome have to be transferred to a special unit for this severe neurological state.

Summarizing:

Patients with severest brain injury due to a primary damage of the upper brainstem or after a tentorial herniation initiated by a supratentorial traumatic process need a well-organized care starting on the site of the accident, followed by management in an intensive care unit experienced in brain trauma patients. A neurological intensive care unit is unit of choice. Neurology has to train experts in neurotraumatology to enable them to come to a clear diagnosis of the different forms of traumatic brain injury and that they know the special obligations in intensive treatment of brain trauma patients. A well-organized system prevents that these patients, who are mostly young people, remain handicapped for their whole life.

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PROGRAMME

STROKE

Pavel Kalvach, Prague, Czech Republic: The cascade of tissue deterioration after an ischemic event

Michael O´Brien, London, United Kingdom: Ischaemic cerebral oedema

Alexander Tsiskaridze, Tbilisi, Georgia: Stroke in Georgia

EPILEPSY

Michael O´Brien, London, United Kingdom: Special problems of women with epilepsy

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Johan A. Aarli, Bergen, Norway: Epilepsy and the immune system

Bernt Engelsen, Bergen, Norway: Treatment of epilepsy

NEUROLOGICAL CRITICAL CARE

Johan A. Aarli, Bergen, Norway: Critical care in acute polyneuropathy

Johan A. Aarli, Bergen, Norway: Critical care polyneuropathy

Franz Gerstenbrand, Vienna, Austria: Critical care in CNS trauma

Lutz Harms, Berlin, Germany: Critical care in CNS infections

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Traumatic brain injury (TBI)

- is a frequent cause of morbidity and mortality in the European countries.
- Incidence between 229 and 1.967 for 100.000 inhabitants
- Highest incidence in men between 15 and 24 years
- Most frequent cause of death for humans under 45 years

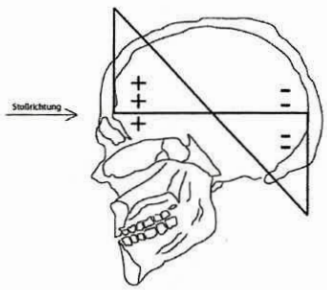
Biomechanics of TBI

- Closed cerebral trauma sometimes combined with fracture of skull
- Open brain injury by a penetrating object (bullet, etc.)
- Two physical factors are important:
  speed v
  acceleration b

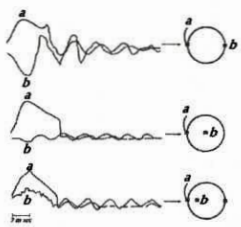
$$b = v^2 / 2s$$

where s is the deceleration distance

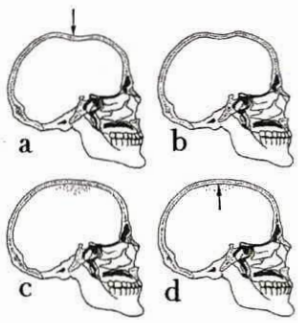
Biomechanics / 2



- Positive pressure at the impact pole
- Negative pressure at the counter pole



Biomechanics / 3

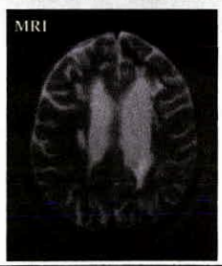


Damage on the counter pole:
Negative pressure causes tissue damage due to gas bubbles (gas, solved in tissue under normal pressure)

Damage on the impact pole:
a) Direct damage due to contact of the skull bone leads to lesions on the surface
b) Due to snapping back of the elastic skull bone negative pressure emerges gas bubbles

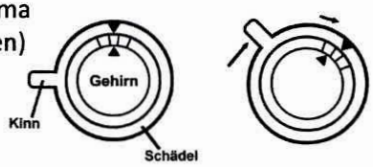
Biomechanics / 4

- Lesions on the surface of the brain



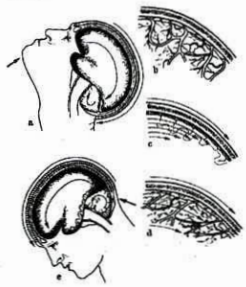
Biomechanics / 5

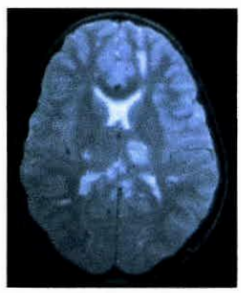
- In linear brain injury (Grcevic) either acceleration or deceleration trauma
- damage on brain tissue depends on localization, intensity, direction of impact
- Rotational trauma (Pudenz-Shelden)



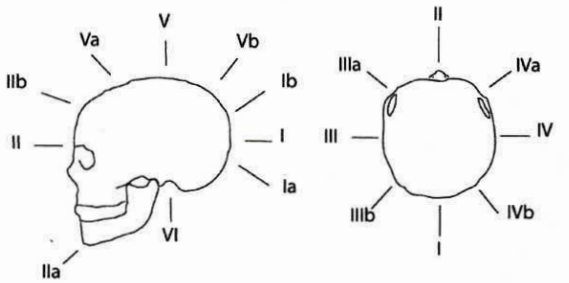
Biomechanics / 6

- Rotational trauma



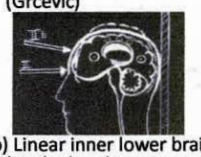


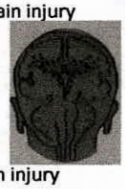
Documentation after Spatz Innsbruck modified



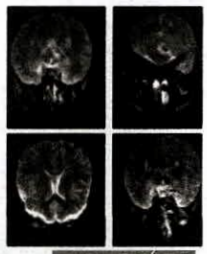
Linear brain injury

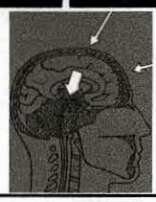
- Linear outer brain injury
  Lesions on brain surface depend on direction and the intensity, contusion zones
- Linear inner brain injury
  a) Linear inner upper brain injury (Grcevic)





  b) Linear inner lower brain injury (Lindenberg)

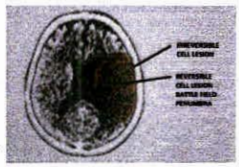




Patterns of cerebral trauma Acceleration - Deceleration

- Outer brain injury
  Coup – side of the impact
  Counter coup – opposite of the impact
- Inner brain injury
  a) Inner upper brain injury – corpus callosum, septum pellucidum, fornix, thalamus, hypothalamus, cingulum
  b) Inner lower brain injury – midbrain (substantia nigra, upper pons), perirubral zone, crura cerebri, tegmentum, periaqueductal gray, perihippocampus, uncus amygdalae, cerebellum
- Rotational brain injury
  laceration (capsula int., basal ganglia), extracerebral hematoma

Different forms of traumatic lesions



- Primary lesions (irreversible)
- Secondary lesions (therapeutic battle field)
  Penumbra, postedemic (diffuse/local), posthypoxic, posthypoxemic (diffuse/local)
- Tertiary lesions (malnutrition, malabsorption, avitaminosis, bed rest syndrome, etc.)
  Encephalopathy, myelopathy, pontine myelinolyse, polyneuropathy
- Quartary lesions
  hydrocephalus occlusus, meningoencephalitis, brain abscess
- Complications
  joint contraction, periarticular ossification, decubitus, pressure lesion of peripheral nerves

Classification of brain injury

- Mild (brain commotion, commotio cerebri)
  Glasgow coma scale (GCS) = 13 – 15
- Moderate (brain contusion, contusio cerebri of mild degree)
  GCS = 9 – 12
- Severe (brain contusion, contusio cerebri of severe degree)
  GCS = 5 – 8
- Severest (brain stem symptoms)
  GCS < 5

Severest brain injury

Primary etiology

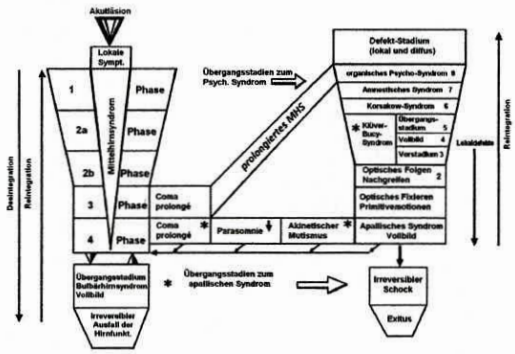
- Primary: Direct lesion of the upper brain stem, linear inner lower brain injury (Lindenberg), impact Type V, Va
- Clinical symptoms: Acute midbrain syndrome
- In some cases developing of acute bulbar brain syndrome
- Bad prognosis, apallic syndrom, brain death

Severest brain injury

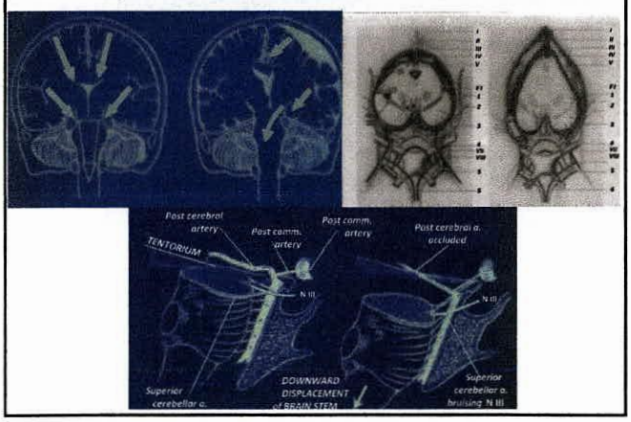
Secondary etiology

- Due to tentorial herniation (central, uncal)
- Consequence of an increased supratentorial pressure (brain edema, extra-, intracerebral haematoma)
- Symptoms of an acute midbrain syndrome
  Development in 5 phases – central herniation
  Development in 2 phases – uncal herniation – transfer in phase 5 of central herniation
- In some cases acute bulbar brain syndrome develops
- Direct remission or transfer in apallic syndrome

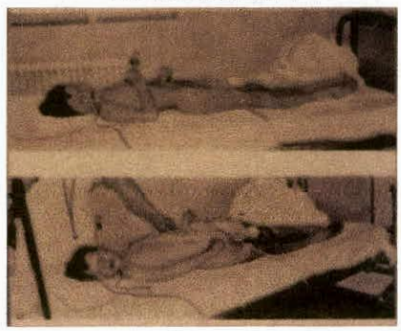
Development of secondary acute midbrain syndrome, further course



Supratentorial volume increase

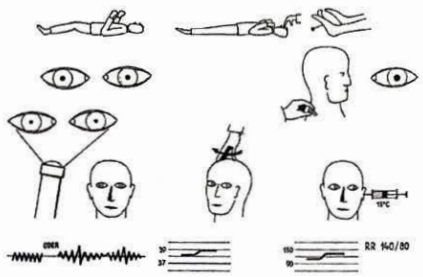


Acute secondary midbrain syndrome
Traumatic brain injury



Phase III
Phase IV

MBS III



Akutes traumatisches Mittelhirnsyndrom - Phase 3:
Schematische Darstellung. Nähere Erläuterung s. Text und Phase 1.
Aus: F. Gerstenbrand und C. H. Lücking.
Die akuten traumatischen Hirnstammschäden, 1970

Phase III, Stretch position, disinhibition of vegetative system

Midbrain syndrome phase IV

- Coma
- Missing blink reflex and ocular movements
- Divergent position of bulbi
- Pupils reduced reaction to light
- Vestibuloocular reflexes disturbed
- Stretch position of the extremities
- Increased muscle tone, pyramidal signs
- Respiration – machine like rhythmus
- Hyperthermia, tachycardia, increased blood pressure

Hirnstammsyndrom: Synopsis

STADIEN DER HIRNSTAMMSCHÄDEN NACH SUPRATENTORIELLER RAUMFORDERUNG ZENTR. HERNIATION		MHS					BHS	
		I	II A	II B	III	IV	I	II
VIGILITÄT		SOMNOLENZ	SOPOR	COMA	COMA	COMA	COMA	COMA
REAKTION	AKUSTISCHE REIZE	GERING VERZÖGERT MIT ZUWENDUNG	VERZÖGERT OHNE ZUWENDUNG	FEHLEND	FEHLEND	FEHLEND	FEHLEND	FEHLEND
	SCHMERZREIZE	PROMPT GERICHTETE ABWEHR	VERZÖGERT UNGERICHTETE ABWEHR	RESTE UNGERICHTETER ABWEHR	BEUGE-STRECK-STELLUNG	STRECK-SYNERGISMEN	REST-STRECK-SYNERGISM.	FEHLEND
OPTOMOTORIK	BULBUS -STELLUNG	NORMAL	NORMAL	BEGINNENDE DIVERGENZ	DIVERGENZ	DIVERGENZ	DIVERGENZ FIXIERT	DIVERGENZ FIXIERT
	BULBUS -BEWEGUNG	PENDELND	SCHWIMMEND	DYSKUNUGIERT	FEHLEND	FEHLEND	FEHLEND	FEHLEND
	PUPILLENWEITE							
	LICHTREAKTION							
KÖRPERMOTORIK	KÖRPERHALTUNG							
	SPONTANMOTORIK	MASSEN- UND WÄLZ-BEWEGUNGEN	MASSENBEWEG. ARME STRECKBEWEG. BEINE	MASSENBEWEG. ARME STRECKBEWEG. BEINE	BEUGE-STRECK-HALTUNG	STRECK-HALTUNG	REST-; NACH-STRECK-HALTUNG	SCHLAFFE HALTUNG
	TONUS	NORMAL	BEINE GERING ERHÖHT	BEINE ERHÖHT	ERHÖHT	STARK ERHÖHT	GERING ERHÖHT	SCHLAFF
	BABINSKI PHÄNOMEN	↑↓	↑↓	↑↓	↑↑	↑↑	↑↑	—
OBLIGAT	ATMUNG							—
VEGETATIV	PULS	LEICHT ERHÖHT	NORMAL	BESCHLEUNIGT	BESCHLEUNIGT	STARK BESCHLEUNIGT	BESCHLEUNIGT	VERLANGSAMT
NICHT OBLIGAT	RR	NORMAL	NORMAL	NORMAL	LEICHT ERHÖHT	ERHÖHT	NORMAL	ERNIEDRIGT
	KÖRPERTEMPERATUR	NORMAL	NORMAL	LEICHT ERHÖHT	ERHÖHT	STARK ERHÖHT	ERHÖHT	NORMAL ERNIEDRIGT

Management of severest traumatic brain injury

4 Phases

- Preclinical management – place of accident
- Immediate measurement in the admitting hospital, with decision for a transfer in the intensive care unit (ICU)
- First measurements in the ICU
- Monitoring and ICU treatment

Preclinical management

- Care for vital function
  Respiration (orotracheal intubation if necessary)
  Blood circulation (infusion)
- Documentation of the impact to the brain
- Registration of side injuries

Management in the admitting hospital

- Control of the vital function
  Artificial respiration if necessary
  Support of blood circulation (infusion, medication)
- Treatment of brain edema
- Neurological status
- Cerebral CT
- X-Ray of cervical spine, skull
- Decision to transfer the patient in the ICU

First measurements in the ICU

- Analgesic sedation (midbrain syndrome)
- Care for vital function
- Intubation
- Central venous catheter
- Bladder catheter
- Treatment of brain edema
- Control of cCT
- If possible cMRI
- ICP-measurement

Treatment of brain edema

- Osmotic therapy
- Diuretic therapy
- Barbiturate
- Hyperventilation

It is still unclear, whether medical therapy directed at lowering ICP offer any benefit to patients outcome (Frank, Neurology 1995)

Special methods

- Craniotomy (both sides)
  In cases with progress
- Hypothermia (mild – 32° - 34°)
- Begin of rehabilitation methods
- Neurosurgical methods

European Federation of
Neurological Societies

Teaching Course in Neurology

Tbilisi, Georgia, September 27-28, 2002

Critical care in CNS trauma

Prof. Franz Gerstenbrand
Ludwig Boltzmann-Institut for Restaurative
Neurology and Neuromodulation
Vienna, Austria

Hall "Narikala 2"
Sheraton Metechi Palace,
Saturday, September 28, 11:20-12:00