63.5%. Only 258 patients (= 3.8%) received neurological- neurosurgical rehabilitation (73% male), 68% within one month after injury, 5% were <16 years, 25% >65 years. Early rehabilitation of 100 patients (= 39%), one fifth referred within first week. Outcome end of "B": GOS 1 = 4%; GOS 2 = 2.7%, GOS 3 = 37.3%, GOS 4 = 26.7%, GOS 5 = 29.3% and end of rehab "B – E" GOS 1 = 1.2%, 2 = 1.7%, 3 = 21.8%, 4 = 36.2%, and 5 = 39.1.

Conclusion. Data on epidemiology and quality management of early functional rehabilitation met the criterion set in 1993 (Ortega-Suhrkamp and von Wild, 2001). Key issue is a multidisciplinary approach for early posttraumatic neurorehabilitation in neurosurgery and neurology. Management of frequent multiple organ lesions and complications (= 57%) without referring the patient to another hospital and early functional outcome confirm the authors concept of neurosurgical early rehabilitation (von Wild Klaus, 2005).

References

Ortega-Suhrkamp E, von Wild KRH (2001) Standards of neurologic-neurosurgical early rehabilitation. Acta Neurochir Suppl 79: 11–19

von Wild Klaus RH (2005) In cooperation with the TBI study council Neurorehabilitation following craniocerebral trauma. Eur J Trauma 4: 344–358

DHT-03-03

Biomechanic aspects in relation to the classification of traumatic brain injury

W. Struhal*, B. Hess, F. Gerstenbrand

* Linz, Austria

Traumatic Brain Injury (TBI) as an attribute to the modern way of life is the most frequent cause of death in the male population aged between 20 and 35 worldwide. Severity and localization of the brain lesion in TBI patients depends on direction, focus and intensity of the impact. A great percentage of brain injury patients suffer from more than one impact. For documentation, archiving and for the biomechanical re-construction of the impact force, the Innsbruck Impact Scheme (IIS), modified after SPATZ is essential. Type I–VI are differentiated. Based on clinical symptomatology, the neuropathological findings and modern neuro-imaging methods supported by biochemical analysis (living pathology – Grcevic), there are three forms of TBI to be differentiated: 1. The linear outer brain trauma (type I–IV) with lesions of the surface of the brain in the contre coup and coup region 2. The linear inner brain trauma, divided in two forms. The linear inner upper brain trauma (type Ia, II, IIb) with periventricular lesions (butterfly defect – Grcevic) The linear inner lower brain trauma (type V, Va) with lesions in the upper brainstem and in the surrounding region (Lindenberg) 3. The rotational brain trauma (type IIIa, b, IVa, b) with intracerebral haematoma, delaceration, extracerebral haematoma (Pudenz, Shelden). Depending to the clinical symptoms, the course of TBI and the changes of the brain tissue, mild, moderate, severe, severest TBI have to be classified. Regarding histological and the anatomical features after TBI there are four well-defined forms of traumatic brain tissue lesions: The primary brain damages occur in the very moment of the force impact to the brain and is an irreversible lesion. Sequences of circulatory and metabolic deficits in the penumbra of primary defect causing local tissue damage, hypoxia and/or hypoxemia are responsible for diffuse and regional secondary tissue lesions. Tertiary lesions developing mostly in a longer posttraumatic course are responsible for encephalopathia, pontine myelinolysis, myelopathia and polyneuropathia, originated by malnutrition, malabsorption, avitaminosis and the bed rest syndrome. Quarterly lesions might emerge months to years after the TBI event in form of hydrocephalus occlusus, meningoencephalitis and brain abscess. As complications contractions of the bigger joints, periarticular ossification, decubitus and lesions of peripheral nerves have to be kept in mind. In the acute state every patient with a TBI needs exact neurological examinations. Different additional explorations depending on the severity grade are necessary, but obligatory in severe and severest conditions. Brain oedema and intracranial haematoma are most endangering the outcome of TBI patients. The increase the intracranial brain volume results in a tentorial hemiation, sometimes followed with foraminal herniation accompanied by an acute mid brain and bulbar brain syndrome (Gersten-brand, Lücking). In severest conditions a traumatic apallic syndrome may develop (Gerstenbrand). It is important not to miss an accompanying cervical spine injury in cases of spinal cord trauma. Regardless on the grade of TBI every patient needs acute therapy. The treatment of TBI has to start already on the site of accident. Severest TBI patients have to be transferred immediately to an up-to-date ICU. Every patient with TBI – regardless to severity – needs a neurorehabilitation program with an individual schedule. A special center with trained personal under the responsibility of a neurologist is necessary. The neurorehabilitation has to start immediately, already in the admitting hospital. A consistent program of modern neurorehabilitation can reduce not only distress and the independency of young patients, it reduces the expenses of the health system to a considerable rate.

DHT-03-04

Late consequences of neurotrauma

A.-L. Siren*

* Germany

Objective. Traumatic brain injury is a leading cause of death and disability for young persons in developed countries, resulting in persistent cognitive dysfunction and behavioral deficits. The immature brain may be particularly vulnerable to injury during critical periods of development. Brain injury at this vulnerable period might lead to exaggerated loss of synapses and unfavorable metabolic alterations which set off the late cognitive decline and brain atrophy. The issue addressed here is (i) whether or not a small localized lesion in the developing parietal cortex is sufficient to trigger global morphological changes in the mature brain, (ii) what the behavioral consequences of such a cascade might be.

Methods. Juvenile (4 week-old) mice were given a unilateral cryolesion of the right parietal cortex. High-resolution three-dimensional magnetic resonance imaging (MRI) and behavioral testing (hole board, elevated plus-maze, Rotarod, open-field, prepulse inhibition of startle, Morris water maze) were performed 3, 6 and 9 months after lesioning.

Results. Significant progressive reduction in brain volume and ventricular enlargement by in vivo 3D MRI were evident at 3, 6 and 9 months after unilateral parietal cortical lesion. This brain atrophy was accompanied with distinct behavioral alterations and spatial learning deficits.

Conclusion. Lesion to the maturing parietal cortex of juvenile mice is by itself the primary cause of a global neurodegeneration, with significant changes in brain morphology and function upon long-term follow-up.

Sunday, 3 June 2007 16.30–18.00 h Salon Oegg

DHT-04

Markers in movement disorders

Chairperson: Heinz Reichmann, Dresden, Germany

DHT-04-01

Abnormal cerebral networks in dystonia

K. L. Leenders*

* Groningen, The Netherlands

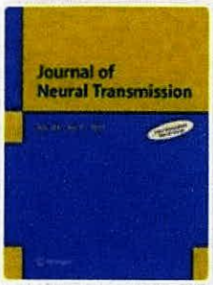
Objective. Idiopathic dystonia comprises various brain disorders in which apparently no structural lesion in the brain is found. The clinical phenomenon of dystonia is thought to be the result of abnormal function of certain cerebral neuronal circuits. It remains however unclear through what abnormal configuration of neuronal circuits dystonia actually occurs.

Methods. Here, first an overview will be given of the studies performed to date in dystonia using activation paradigms and neuroimaging in vivo in dystonia patients. Next, the recent studies of our own group using motor execution and motor imagining paradigms in a 3T fMRI setup in healthy controls and in patients with cervical dystonia, dystonia in complex regional pain syndrome-I will be presented. Also the problem of initiation and inhibition of movements will be discussed showing the results in a group of healthy volunteers. First results will be shown applying interleaved TMS in a 3T fMRI setting.

Results. In healthy volunteers, imagery activated bilaterally the superior and inferior parietal cortex, prefrontal cortex, cerebellum, left premotor cortex and supplementary motor area (SMA). Motor execution additionally activated left primary motor cortex and showed less activation in the premotor and prefrontal cortex. Cervical dystonia patients showed reduced activation in the parietal cortex,

Journal of Neural Transmission

Translational Neuroscience, Neurology and Preclinical Neurological Studies, Psychiatry and Preclinical Psychiatric Studies



Volume 114, issue 7, July 2007

13 articles in this issue

The other-race effect for face perception: an event-related potential study

M. J. Herrmann, T. Schreppel ... A. J. Fallgatter

OriginalPaper | Published: 23 February 2007 | Article: 951

Comparison of cognitive functions between people with silent and wild-type butyrylcholinesterase

I. Manoharan, A. Kuznetsova ... S. Darvesh

OriginalPaper | Published: 22 February 2007 | Article: 939

Abstracts – 39th International Danube Symposium for Neurological Sciences and Continuing Education and 1st International Congress on ADHD, from childhood to adult disease

Abstract | Published: 01 July 2007 | Pages: XLIII - CXLI

39th International Danube Symposium
for Neurological Sciences and Continuing Education
in conjunction with the
1st International Congress on ADHS
from childhood to adult disease

Biomechanic aspects in relation to the classification of traumatic brain injury

W. Struhal[1], B. Hess[2], F. Gerstenbrand[2]

1) Neurological Intensive Care Unit, AKH, Linz
2) Ludwig Boltzmann Institute for Restorative Neurology, Vienna

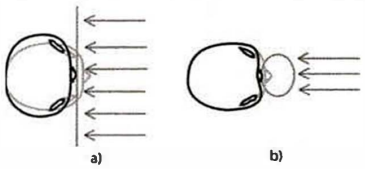
walter.struhal@akh.linz.at

Traumatic brain injury (TBI)

- incidence between 229 and 1.967 for 100.000 EU inhabitants
- highest incidence in men between 15 and 24 years
- most frequent cause of death for humans under 45 years

Different types of TBI

- Closed cerebral trauma
  sometimes combined with fracture of skull



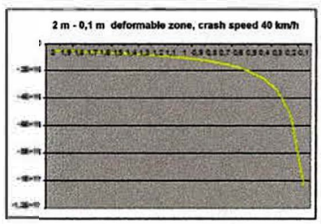
- Open brain trauma by a penetrating object (bullet, etc.)

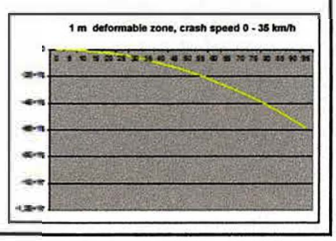
Biomechanics of TBI

- Two physical factors are important:
  speed *v (m/s)*
  acceleration *b (m/s²)*
  acceleration distance *s (m)*

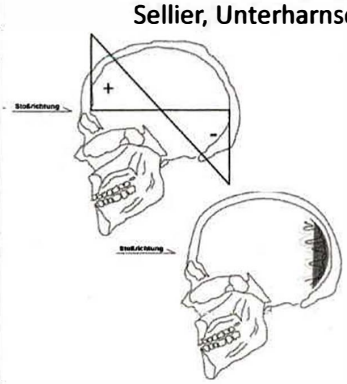
$$b = \frac{-v^2}{2s}$$

In fact it's $b = \frac{v_e^2 - v_0^2}{2s}$, but v_e^2 is regarded to be 0 = deceleration until complete stop





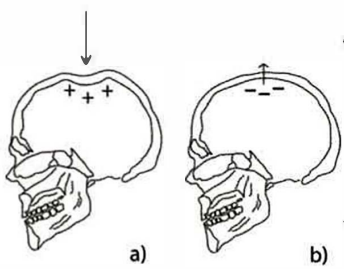
Biomechanics, physical analysis
Sellier, Unterharnscheidt, 1963



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

with permission of K. Jellinger, Vienna

Biomechanics, cavitation trauma

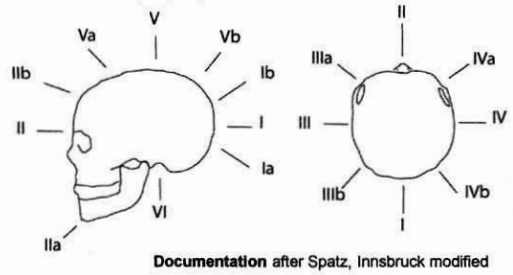


- Lesions on the impact region (a):
  Direct damage due to impressed skull bone leads to lesions on the brain surface, cortical region, overpressure
- Due to snapping back of the elastic skull bone (b), negative pressure emerges cortical lesions

Aetiology of brain tissue damage after closed skull trauma – impact scheme

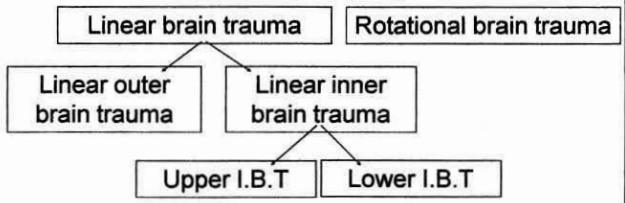
Brain tissue damage depends on
- Direction, form of impact
- Location of impact
- Intensity of the force

Multiple impacts possible



Documentation after Spatz, Innsbruck modified

Different Types of Brain Trauma Classification by biomechanical analysis



Biomechanics acceleration, deceleration impact

- Linear brain trauma (Grcevic, Lindenberg)
- Rotational trauma (Pudenz-Shelden)

Damage on brain tissue depends on localisation, direction, intensity of impact.

Patterns of cerebral trauma

Acceleration – Deceleration trauma

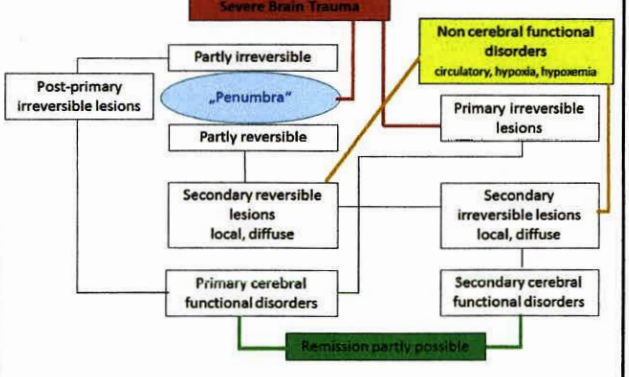
- Linear brain injury
  - Outer brain injury
    1. Coup - local lesions on the impact region
    2. Countre coup – opposite to the impact
  - Inner brain injury
    1. Inner upper brain injury – lesions: corpus callosum, septum pellucidum, fornix, thalamus, hypothalamus, cingulum
    2. Inner lower brain injury – midbrain (substantia nigra, perirubral zone, crura cerebri, tegmentum, periaqueductal gray, upper pons), perihippocampus, uncus amygdalae, cerebellum
- Rotational brain trauma
  1. Laceration (capsula int., basal ganglia)
  2. Intracerebral haemorrhage (thalamus, hypothalamus)
  3. Extracerebral haematoma (subdural, epidural)

Type of Traumatic Brain Damage

- Primary lesions, immediately by impact, mostly irreversible
  - Outer brain trauma
  - Inner brain trauma
  - Rotational brain trauma
- Secondary lesions
  1) primary impact, Umbra/Penumbra
     - regional lesions
  2) Non-cerebral functional disorders, hypoxia, hypoxemia, circulatory disturbances
     - local, regional, diffuse lesions
  3) Tentorial herniation
     a) local pressure, tentorial edge
     - local lesions (upper brain stem, medial tentorial region)
     - regional lesions due stenosis of A.cerebri posterior

     b) downwards displacement of brain stem
     - local lesions due arterial and venous stenosis
     - brain nerve lesions (N.oculo-motorius)

Schema of brain trauma, primary and secondary lesions, Grcevic, Gerstenbrand

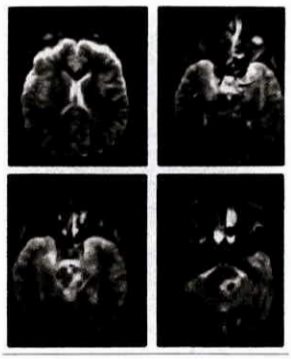


Linear Outer Brain Trauma (Type I, II, III, IV)

- Coup lesions, contre-coup lesions
  - Cortical, sub-cortical, meningeal damage, funnel-shaped
  - Type II minor lesions frontal
    absorption of forces by facial skeleton
  - Type I severe lesions fronto-temporal
    Countre-coup
  - Type III, IV mostly combined with rotational brain trauma

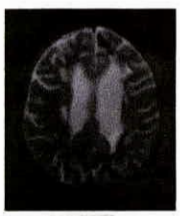
Linear outer brain trauma

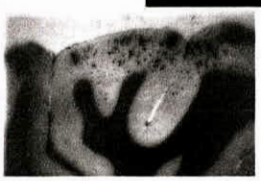
Lesions on brain surface depend on direction and the intensity, as well as on the contusion zones



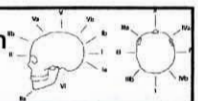
Linear outer brain injury

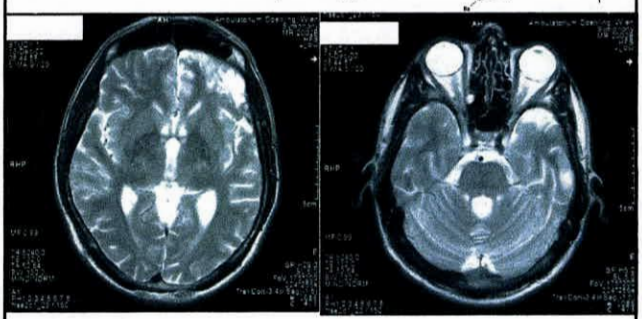
- Lesions on the surface of the brain (cortical-subcortical, funnel-shaped)





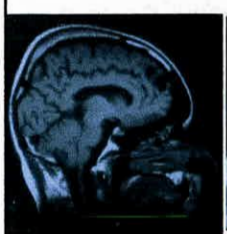
Linear outer brain trauma impact type I

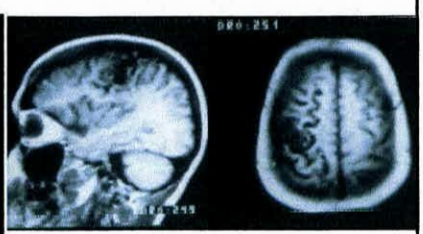




Severe lesions frontal, temporal, minor lesion cerebellar

Different Types of Linear Outer Brain Trauma





Impact type I

Impact type IV

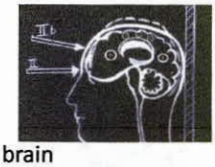
Linear Inner Brain Trauma

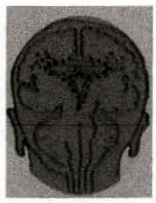
Primary Lesions
Grcevic, Lindenberg

- Inner upper brain trauma (Grcevic)
  - Lesions peri-ventricular (butterfly type): corpus callosum, septum pellucidum, fornix, thalamus, hypothalamus, cingulum
- Inner lower brain trauma (Lindenberg)
  - midbrain-pons lesions (substantia nigra, perirubral zone, crura cerebri, tegmentum, periaqueductal gray, upper pons),
  - surrounding brain regions (perihippocampus, uncus amygdalae, cerebellum)

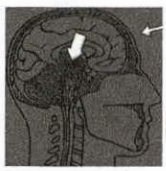
Linear Inner Brain Trauma

a) Linear inner upper brain trauma (Grcevic) butterfly lesions
Type IIb, Ia (II)





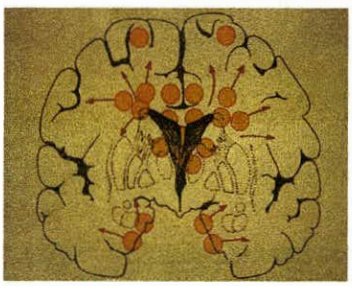
b) Linear inner lower brain trauma (Lindenberg) lesions brain stem, surrounding brain region
Type V, Va



Linear Inner Upper Brain Trauma (GRCEVIC) Type IIb, Ia (II)

- Lesions in the centro-axial brain region, butterfly type:
  - most frequently:
    - corpus callosum
    - septum pellucidum
    - peri/-paraventricular zone
    - thalamus
  - partly:
    - hippocampal area
    - upper brain stem
    - parasagittal region
    - hypothalamus

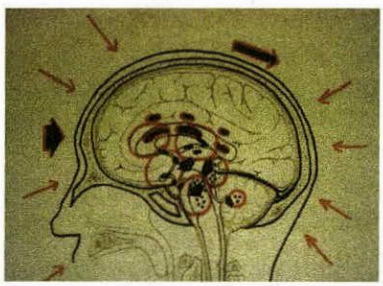
Linear Inner Upper Brain Trauma Schematic drawing (N. Grcevic)



Impact type IIb, Ia, (II)

Main lesions, periventricular

Linear Inner Upper Brain Trauma Schematic drawing (N. Grcevic)

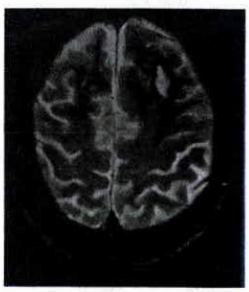


Impact type II, IIa, often with rotational component

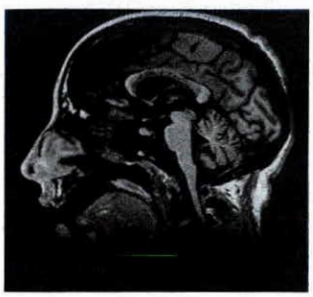
Lesions, periventricular, upper brain stem

Boxing impact frontal region

Linear Inner Upper Brain Trauma Type Ib

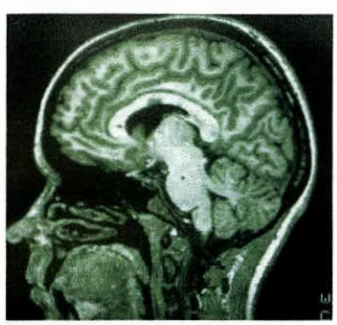


Parasagittal lesion, butterfly type



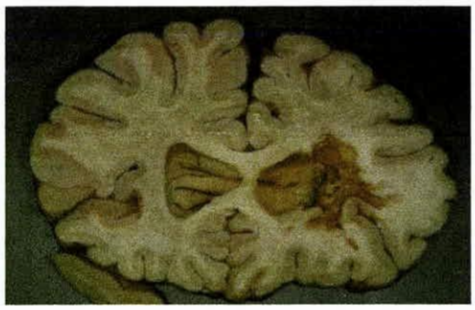
Lesion corpus callosum, butterfly type

Linear Inner Upper Brain Trauma, Impact Type IIb



Local lesion corpus callosum

Linear Inner Upper Brain Trauma Type Ib

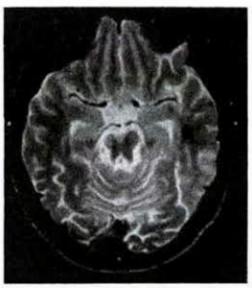


Frontal white matter, periventricular damage

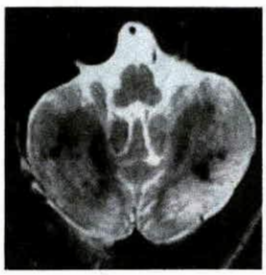
Linear Inner Lower Brain Trauma (Lindenberg) Type V, Va

- Primary lesions
  - upper brain stem
  - surrounding brain region
    - Medial temporal lobe
    - cerebellum
- Secondary lesions: tentorial contusion
  - upper brain stem
  - medial temporal lobe
  - vascular lesions (regional)

Linear Inner Lower Brain Trauma Type Va, Primary lesions

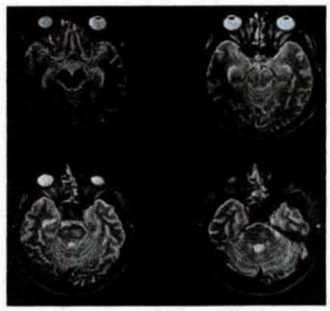


Mesencephalon



Cerebellum

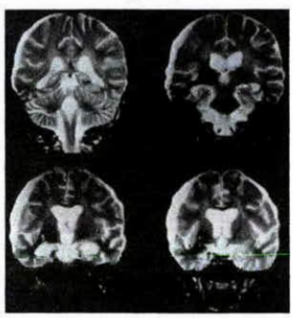
Linear Inner Lower Brain Trauma Type Va, Combination with tentorial herniation



Primary lesion pons,medulla oblongata, (upper part)

Secondary lesion, tentorial herniation lower midbrain

Linear Inner Lower Brain Trauma Combination type Va, IVa



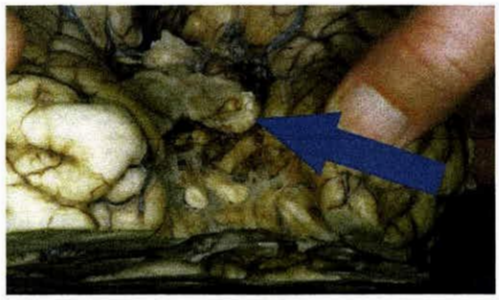
Lesions hippocampal Parahippocampal

Hygrom fronto-parietal left

Linear Inner Lower Brain Trauma, Type Va, Primary lesions

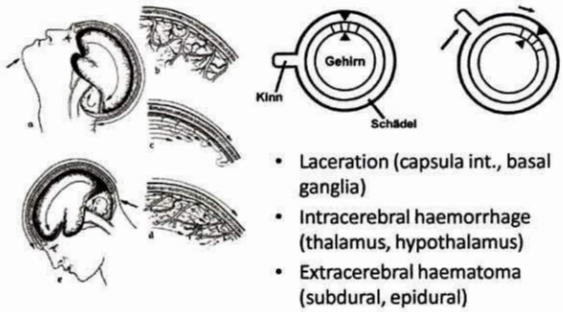
Gliotic lesions with haemosiderin deposition, lower midbrain, pons

Linear Inner Lower Brain Trauma
Combination with uncal tentorial herniation



Primary lesion in the upper mesencephalon, secondary lesion after uncal herniation (arrow)

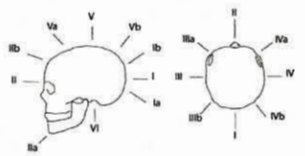
Rotational trauma – Scheme
Pudenz-Shelden



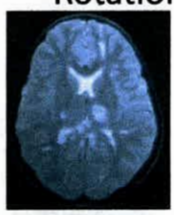
- Laceration (capsula int., basal ganglia)
- Intracerebral haemorrhage (thalamus, hypothalamus)
- Extracerebral haematoma (subdural, epidural)

Rotational Trauma
(Pudenz-Shelden)
Type Ia, Ib, IIa, IIb, IIIa, IIIb, IVa, IVb, VI

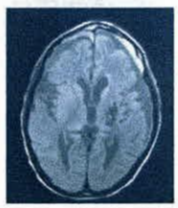
- Intracerebral laceration (basal ganglia, capsula interna)
- Intracerebral hematoma (thalamus, hypothalamus)
- Extracerebral hematoma (subdural, epidural)



Rotational trauma, H.R., 28a



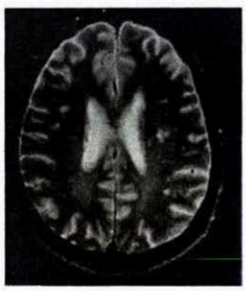
Thalamic haemorrhage both sides
Intracerebral bleedings
Dilaceration intern capsle left



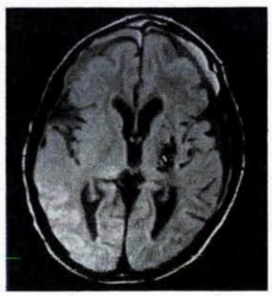
Thalamic lesions left side
Hygroma frontal left side, minimal right
Cortical atrophy, frontal, temporal lobe both sides

Control MRI after 6 months

Rotational Brain Trauma
Type IIb

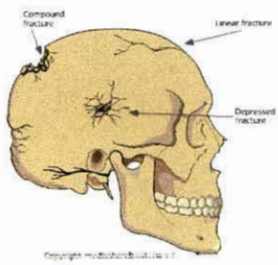


White matter lesions, small haematoma



Lesions:
basalganglia, capsula interna

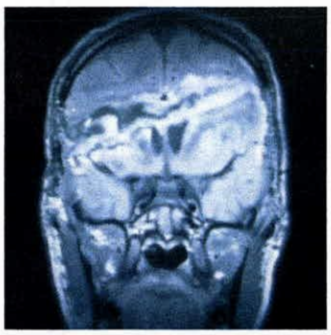
Open Brain Trauma



- Open skull fracture
- Open impression fracture
- Compound skull fracture
- Penetration skull fracture
  - Bullet injury
  - bolt pistols
  - Axe injury

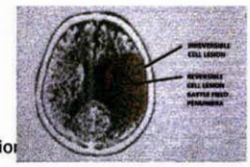
open skull: different influence of the acting force, additional direct lesion

Open Brain Trauma



Bullett injury, suicide, brain death

Different forms of traumatic lesions



- Primary lesions (irreversible)
- Secondary lesions (therapeutic battle field)
  - Penumbra, postedemic (local),
  - posthypoxic, posthypoxemic (diffuse/ local)
- Tertiary lesions (malnutrition, malabsorptio 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

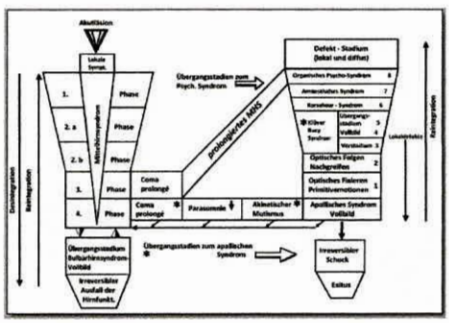
Classification of brain trauma

- Mild traumatic brain injury
  Glasgow Coma Scale (GCS) = 13 – 15
- Moderate traumatic brain injury
  GCS = 9 – 12
- Severe traumatic brain injury
  GCS = 5 – 8
- Severest brain injury – brain stem symptoms (acute midbrain syndrome, bulbar brain syndrome)
  GCS < 5

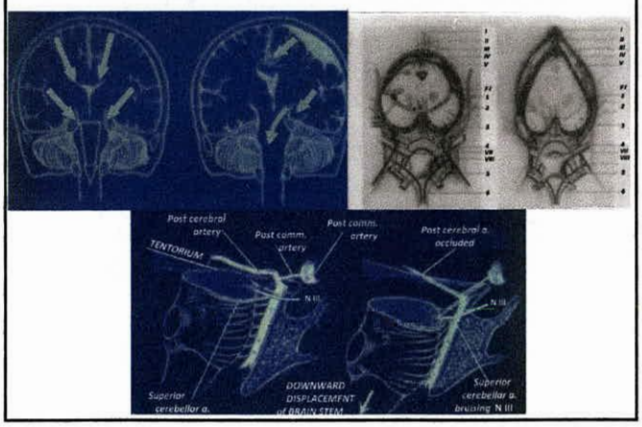
Severe Brain Trauma

further course

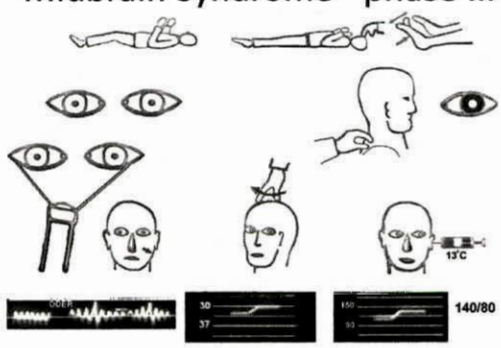
F. Gerstenbrand, 1967, 1977, F. Gerstenbrand, E. Rumpl, 1983



Supratentorial volume increase



Midbrain Syndrome - phase III



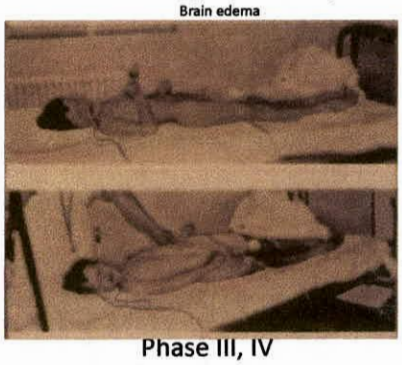
Phase III, Stretch position, disinhibition of vegetative system

Midbrain syndrome phase IV

- Coma
- Blinking reflex missing
- Divergent position of bulbi
- Ocular movements blocked
- Pupils reduced reaction to light
- Ocular cephalic reflex disturbed
- Vestibuloocular reflexes disturbed
- Stretch position of the extremities, trunk
- Increased muscle tone, hyperreflexia, pyramidal signs
- Respiration – machine like rhythm
- Hyperthermia, tachycardia, increased blood pressure

Acute secondary midbrain syndrome

Brain edema



Phase III, IV

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	-STELLUNG BULBUS -BEWEGUNG	NORMAL / PENDELND	NORMAL / SCHWIMMEND	BEGINNENDE DIVERGENZ / DYSKUNJUGIERT	DIVERGENZ / FEHLEND	DIVERGENZ / FEHLEND	DIVERGENZ FIXIERT / FEHLEND	DIVERGENZ FIXIERT / FEHLEND
	PUPILLENWEITE							
	LICHTREAKTION							
KÖRPERMOTORIK	KÖRPERHALTUNG							
	SPONTANMOTORIK	MASSEN- UND WALZBEWEGUNGEN	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

Acute traumatic midbrain syndrome

Primary etiology

- Direct lesion of the upper brain stem (linear inner lower brain injury), impact Type V, Va
- Clinical symptoms: Acute midbrain syndrome, immediately development
- Acute bulbar brain syndrome possible
- Poor prognosis
  apallic syndrom, brain death

Acute Traumatic Midbrain Syndrome

Secondary etiology

- Increased supratentorial pressure (brain edema, extra-, intracerebral haematoma)
- Tentorial herniation (central, uncal)
- Symptoms of an acute midbrain syndrome
  Development in 5 phases – central herniation
  Development in 2 phases – uncal herniation
  Transfer in phase 4 or 5 of central herniation
- Acute bulbar brain syndrome possible
- Direct remission possible
- Transmission to apallic syndrome possible

Management of Severest Brain Trauma

4 Phases

- Preclinical management – on the site of accident
- Immediate measurement in the admitting hospital
- Decision for a transfer to the intensive care unit (ICU)
- First measurements in the ICU

Preclinical Management

- if GCS, always with pupil status
- Care for vital function, respiration
  - orotracheal intubation, if necessary (never nasal, without exact knowledge of bone injuries)
  - Stabilization of blood circulation (infusion)
  - if necessary feeding tube to empty stomach (again only oral, never nasal)
- Documentation of the impact (Spatz – Innsbruck Scheme)
- Registration of secondary injuries

Hospital management

- Neurological status
- Control of the vital function
  - intubation and artificial respiration if necessary
  - support of blood circulation (infusion, medication)
- Treatment of brain edema
  - hyperventilation
  - cooling
  - medication
- fast Spiral CT/ Multislice CT brain/skull+neck+thorax (not state of the art and only if CT scanner is not avaliable: X-Ray of cervical spine, skull)
- Neurosurgical control
- Decision to transfer the patient to the ICU

First Measurements ICU

- Care for vital function
- Central venous catheter
- Bladder catheter
- Analgosedation (acute midbrain syndrome, obligatory)
- Treatment of brain edema
- control of cMRI (CT might not detect diffuse axonal damage)
- parenchymatous ICP-measurement if necessary (cave: complications)

Apallic Syndrome (AS), after acute severe brain trauma

- Initial stage:
  - acute midbrain syndrome (central 5 phases, lateral 2 phases – transmission in phase 4, 5)
  - acute bulbar brain syndrome (2 phases)
- Transition stage to AS (3 phases)
- Full stage of AS
- Remission stage (8 phases)
- Defect stage (multilocular lesions, regional lesions, diffuse lesions)

Rehabilitation after traumatic brain injury

- often not only motoric and sensory signs but also neuropsychological deficits as consequence of a frontal, temporal or parietal lesion
- individual therapy is necessary after exact neuropsychological testing, including logopedic, ergotherapeutic and physiotherapeutic as well as cognitotherapeutic strategies
- immediate planning after admission
- reduces distress, independency of patients and expense of health system
- repetitive testing is important to detect tertiary or quartery lesions

Take home messages

- different biomechanic forces on brain tissue produce very distinct lesion patterns dependent on the direction of impact, the force and size of impact
- diagnosis and also prognosis might be simplified by the modern classification in combination with a documentation of
  1. impact direction
  2. linear or rotational trauma