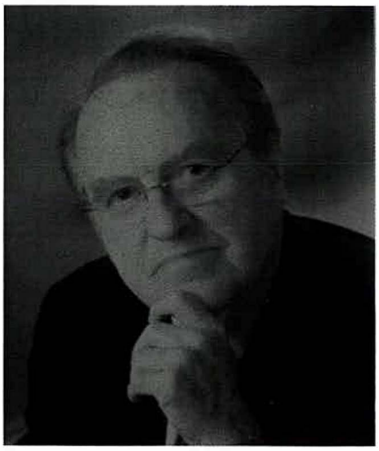

Franz Gerstenbrand,
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Vienna, Austria

TRAUMATIC BRAIN INJURY, BIOMECHANIC ASPECTS AND CLASSIFICATION

Worldwide as an attribute to the modern way of life the increased number of traumatic brain injury (TBI) can be observed. Improved standards of living, traffic, modernisation, industrialization, development and advancement of the society must be accused for this development. Between the age of 20 to 35 the most frequent cause of death is traumatic brain injury.

Depending on the direction and the intensity to the impact of the head an open or a closed cerebral trauma has to be differentiated. Based on clinical symptomatology, neuropathological findings and the results of modern neuro-imaging methods (living pathology-Grcevic) three types of TBI have to be differentiated: The linear outer brain trauma with lesions on the surface of the brain (Coup, Contre Coup), the linear inner brain trauma and the rotational brain trauma. The linear inner brain trauma has to be divided in the linear inner upper brain trauma with lesions around the ventricle system (butterfly type - Grcevic) and the linear inner lower brain trauma (Lindenberg) the most dangerous type of TBI , with lesions in the mid brain region and the surrounding brain areas. The rotational brain trauma causes intracerebral haematoma and dilaceration of brain tissue mostly in the inner capsle and the basal ganglia as well as extracerebral haematomas, intracranial haematomas.
More then one impact with a multifocal influence on the brain is possible.

The documentation of an impact on the head using the Innsbruck Impact Scheme allows to analyze the biomechanical forces on the brain. The localization and the grade of primary traumatic brain defects can be calculated. In the severity of TBI the modern classification divided between mild, moderate, severe and severest TBI.

Regarding the anatomical and histological changements four forms of traumatic lesions of brain tissue can be differentiated. The primary brain lesions resulting at the moment of the impact are irreversible. Secondary brain lesions as sequencies of circulatory deficit in the Penumbra of the primary defect cause local tissue lesions, hypoxia and/or hypoxemia are responsible for diffuse and regional brain tissue lesions.

Tertiary lesions, developing mostly in a longer posttraumatic course are responsible for encephalopathia, pontine myolinolysis, myelopathia and polyneuropathia, originated by malnutrition, malabsorption, avitaminosis and the bed rest syndrome. As quartery lesions developing in posttraumatic stage, sometimes months till years afterwards a hydrocephalus occlusus, mengingo- encephalitis and brain abcess can be observed. 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 controls with different additional examinations depending on the grade, but obligatory in severe and severest conditions. A neuromonitoring is necessary for every patient.

The most dangerous and acute complications of TBI are brain oedema and intracraniel haematoma, both with an increase of the intracranial volume followed by tentorial herniarion, sometimes by foraminal herniation, accompanied by an acute mid brain syndrome and acute bulbar brain syndrome (F.Gerstenbrand, C.H.Lücking). In severe conditions a traumatic apallic syndrome, unfortunately in English literature called vegetative state, can develop (F.Gerstenbrand).
Never to forget is an accompagnied damage of the cervical spine together with a spinal cord trauma.

Every patient with the TBI needs an acute therapy independently to the grade. The treatment programme has to be accompanied by a monitoring system, essentially including MTBI (Vos et al). In severe and severest form of a TBI a neurosurgical consultation is obviously necessary. The treatment of TBI has to start out of the hospital, already on the place of the accident, initiated with measures to care the vital functions. Severest TBI patients have to be transferred immediately in a modern equipped ICU.

Every patient with TBI, independent to the grade of the brain lesions, needs a neurorehabilitation with a consequent programme. A special centre with trained personal under the responsibility of a neurologist is necessary. The neurorehabilitation has to start immediately, already in the admitting hospital. A consequent programme of modern neurorehabilitation can reduce not only suffering and the individuality of young patients, it reduces the expenses of the health system in a considerable rate.

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DANUBIAN NEUROLOGICAL TEACHING COURSE

Conference Room MINCU, Best Western Parc Hotel, Bucharest
ROMANIA

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2007

Traumatic Brain Injury

New aspects in classification, using biomechanical and neuropathological analysis

F. Gerstenbrand, B. Hess, W. Struhal

Danubian Neurological Teaching Course
20-21 March, 2007
Bucharest, Romania

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 (most frequent cause of death between age of 20 – 35 years worldwide in the male population)

Different types of TBI

- Closed Brain Trauma
  sometimes combined with fracture of skull
- Open Brain Trauma by a penetrating object (bullet, etc.)

Biomechanics of TBI

- Two physical factors are important:

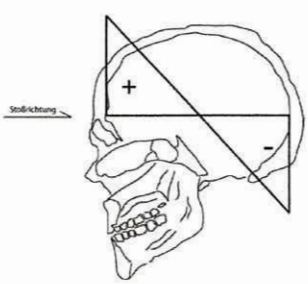
speed v

acceleration b

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

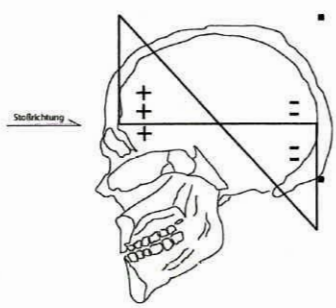
where s is the deceleration distance

Biomechanics, physical analysis
after Sellier and Unterharnscheidt, 1963



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

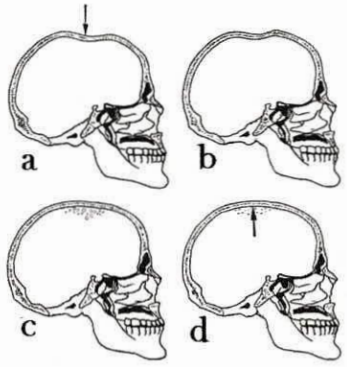
Biomechanics (impact trauma)
after Sellier and Unterharnscheidt, 1963



- Lesions on the counter pole: Negative pressure causes tissue damage due to gas bubles, cortical region (gas solved in tissue under normal pressure)
- Lesions on the impact pole (coup):
  Direct damage due to contact on the skull bone leasd to leasions on the surface, cortical region

Biomechanics, cavitation trauma

after A.G. Gross, 1958



Lesions on the impact region (b):
Direct damage due to impressed skull bone , positive pressure, leads to lesions on the brain surface, cortical region, overpressure

Due to snapping back of the elastic skull bone, negative pressure emerges gas bubbles (d), cortical lesions

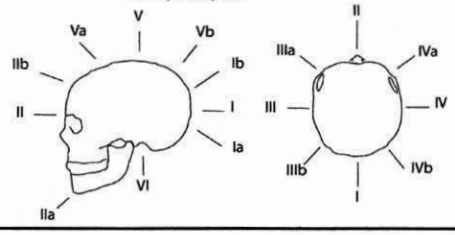
Etiology of brain tissue damage

after closed skull trauma – impact scheme

Documentation after Spatz, Innsbruck modified

Brain tissue damage depends on

- Direction and form of impact
- Location of impact
- Intensity of the force
- Multiple impacts



Biomechanics

- Linear brain injury (Grcevic, Lindenberg) either acceleration or deceleration trauma
  - Damage on brain tissue depends on localisation, intensity, direction of impact
- Rotational trauma (Prudenz-Shelden)

Patterns of cerebral trauma
Acceleration - Deceleration

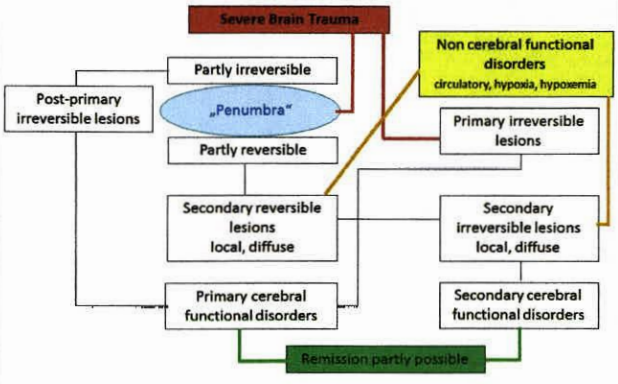
- Linear brain injury
  - Outer brain injury
    1. Coup – local lesions on the impact region
    2. Countre coup – opposite of the impact
  - Inner brain injury
    1. Inner upper brain injury – lesions: corpus callosum, septum pellucidum, fornix, thalamus, hypothalamus, cingulum
    2. Inner lower brain injury – midbrain (substantia nigra, perirubral zone, crura cerebri, tegmentum, periaqueductal gray, upper pons), perihippocampus, uncus amygdalae, cerebellum
- Rotational brain injury
  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



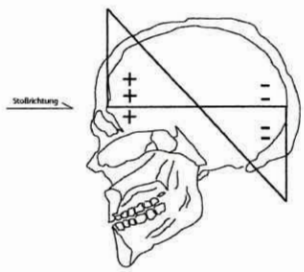
Linear Outer Brain Trauma (Type I, II, III, IV)

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

Linear Outer Brain Trauma

Biomechanics

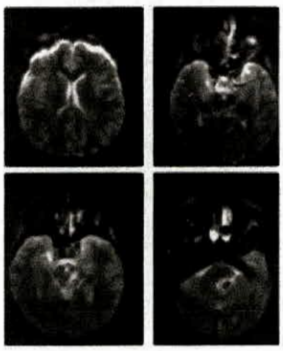
Sellier, Unterharnscheidt, 1963; Grcevic, 1965



- Lesions on the counter pole: Negative pressure causes tissue damage (cortical region) due to gas bubbles, (gas solved in tissue under normal pressure)
- Lesions on the impact pole (coup region):
  Direct damage due contact on the skull bone, positive pressure, leads to lesions on the cortical region

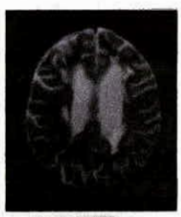
Linear outer brain trauma

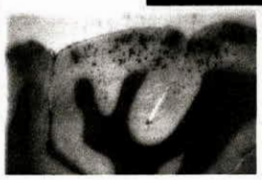
- Lesions on brain surface depending on direction and the intensitiy, as well as contusion zone



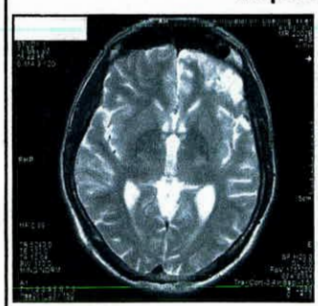
Linear outer brain injury

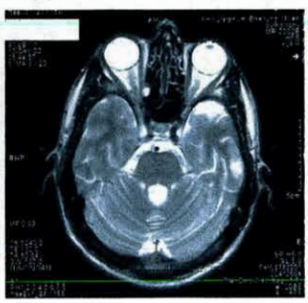
- Lesions on the surface of the brain (cortical-subcortical, funnel-shaped)





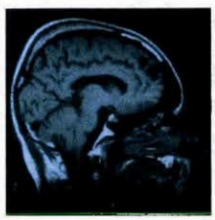
Linear outer brain trauma, impact type I

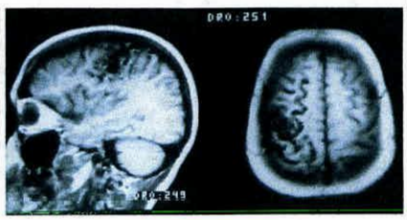




Severe lesions fronto-temporal, minor lesion cerebellar

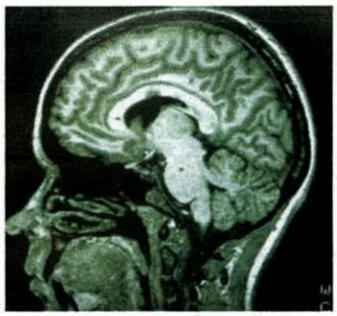
Different types of linear outer brain trauma





Impact type I Impact Type IV

Linear Inner Upper Brain Trauma, Impact Type IIb



Local lesion corpus callosum

Linear Inner Brain Injury

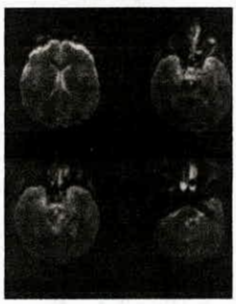
Primary Lesions

Grcevic, Lindenberg

- Inner upper brain trauma (Grcevic)
  - Lesions periventricular (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)

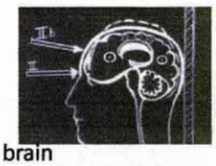
Linear outer brain trauma

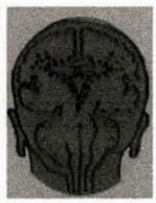
- Lesions on brain surface depend on direction and the intensity, contusion zones



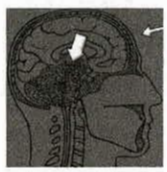
Linear Inner Brain Trauma

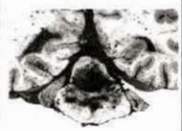
- a) Linear inner upper brain trauma (Grcevic) butterfly lesions
  Type IIb, Ia (II)





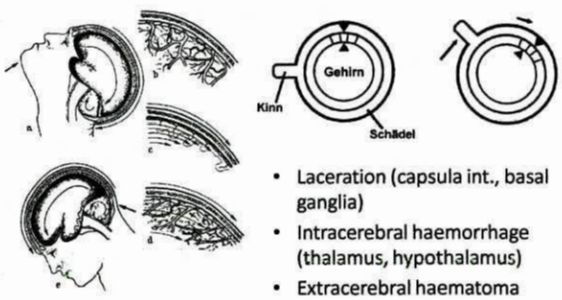
b) Linear inner lower brain trauma (Lindenberg) lesions brain stem, surrounding brain region
Type V, Va





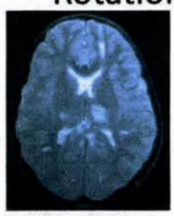
Rotational trauma – Scheme

Pudenz-Shelden

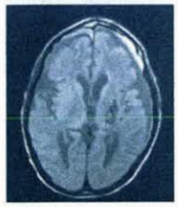


- Laceration (capsula int., basal ganglia)
- Intracerebral haemorrhage (thalamus, hypothalamus)
- Extracerebral haematoma (subdural, epidural)

Rotational trauma, H.R., 28a



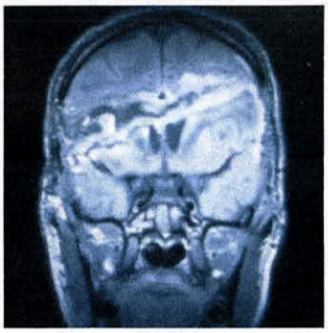
Thalamic haemorrhage both sides
Intracerebral bleedings
Dilaceration intern capsle left



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

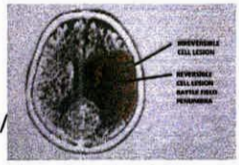
Control MRI after 6 months

Open brain traumatic injury



Bullett injury, suicide, brain death

Different forms of traumatic lesions



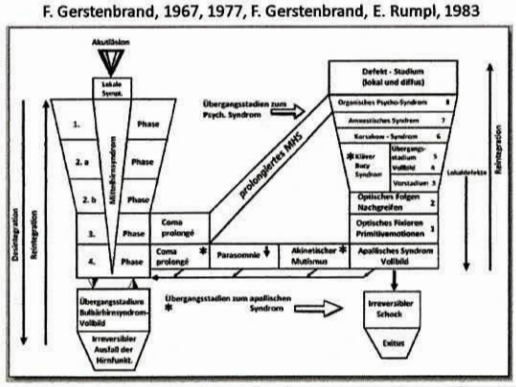
- Primary lesions (irreversible)
- Secondary lesions (therapeutic battle field)
  - Penumbra, postedemic (local),
  - posthypoxic, posthypoxemic (diffuse/ local)
- Tertiary lesions (malnutrition, malabsorption, avitaminosis, bed rest syndrome, etc.)
  Encephalopathy, myelopathy, pontine myelinolyse,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

Classification of brain injury

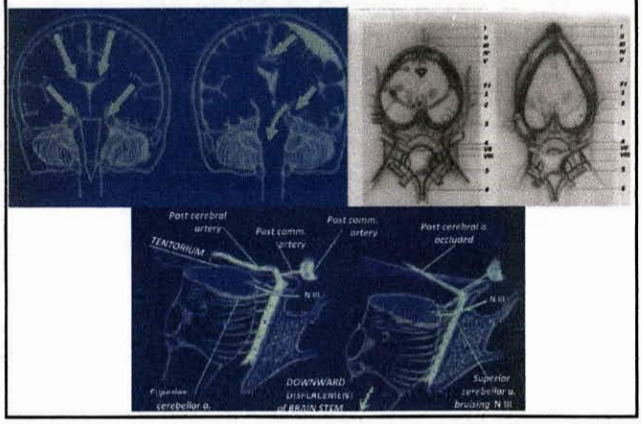
- Mild traumatic brain injury
  (brain commotion, Commotio Cerebri, Hirnerschütterung)
  Glasgow Coma Scale (GCS) = 13 – 15
- Moderate traumatic brain injury
  (brain contusion, Contusio Cerebri – mild degree)
  GCS = 9 – 12
- Severe traumatic brain injury
  (brain contusion, Contusio Cerebri – severe degree)
  GCS = 5 – 8
- Severest brain injury – brain stem symptoms (acute midbrain syndrome, acute bulbar brain syndrome)
  GCS < 5

Secondary acute midbrain syndrome, further course

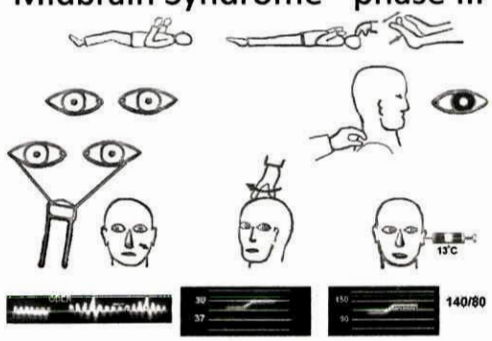
F. Gerstenbrand, 1967, 1977, F. Gerstenbrand, E. Rumpl, 1983



Supratentorial volume increase



Midbrain Syndrome - phase III



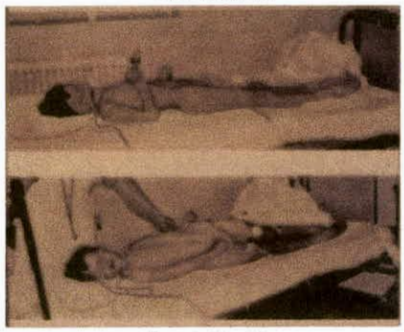
Phase III, Stretch position, disinhibition of vegetative system

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 WÄLZBEWEGUNGEN	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

- Primary direct lesion of the upper brain stem, linear inner lower brain injury (Lindenberg), impact Type V, Va
- Clinical symptoms: Acute midbrain syndrome, immediately development
- In some cases acute bulbar brain syndrome possible
- Poor prognosis
  apallic syndrome, brain death

Acute Traumatic Midbrain Syndrome

Secondary etiology

- Sequence of an 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
- In some cases acute bulbar brain syndrome possible
- Direct remission possible
- Transmission to apallic syndrome possible

Management of severest traumatic brain injury

4 Phases

- Preclinical management – on the site of accident
- Immediate measurement in the admitting hospital,
- 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 secondary injuries

Management By 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
- Neurosurgical methods
- Decision to transfer the patient to the ICU
- Begin of rehabilitation methods

First Measurements in the ICU

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

Treatment of brain edema

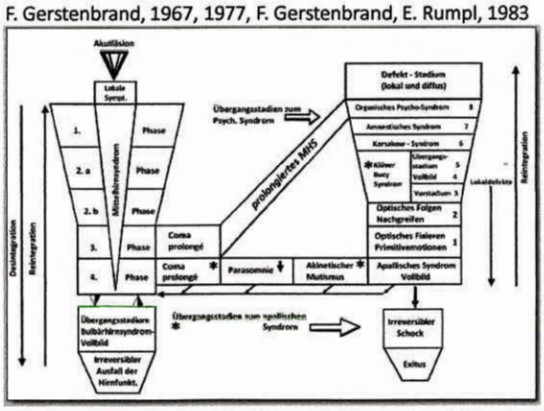
- Osmotic therapy
- Diuretic therapy
- Barbiturate
- Hyperventilation

Special methods in treatment of brain edema

- Hypothermia (mild – 32° - 34°)
- Craniotomy (both sides) in cases with progress

Severest Brain Trauma, further course

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



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)

Apallic syndrome, pat. G.B., 36ª
traumatic brain injury, 1975

No modern treatment

Irreversible tertiary lesions, complications

Exitus after 14 months

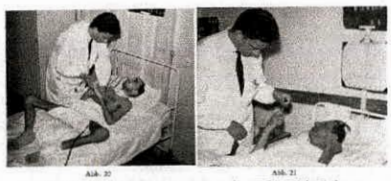
Apallic syndrome, pat. E.S., 19ª
traumatic brain injury, 1992

Modern treatment program in special center for apallic syndrome patients

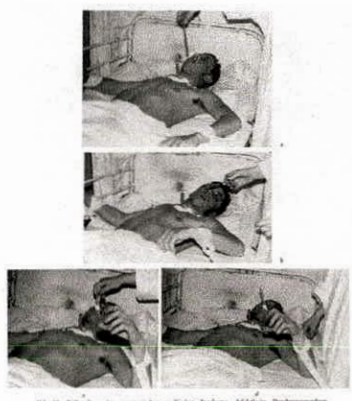
No tertiary lesions, minimal complications

Remission after 5 months to minimal defect state

Traumatic apallic syndrome full stage, primitive motor patterns



- Grasping reflex
  - Fig. 20: tonic grasping
  - Fig. 21: phasic grasping



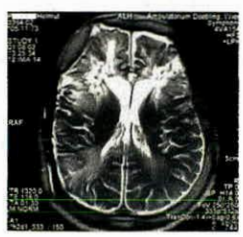
Pat. G.N., 39a

- Traumatic apallic syndrome, full stage
- Optic orale reflex, Bulldog-Reflex

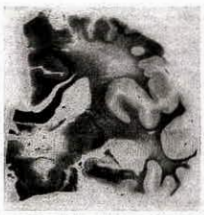
Traumatic Apallic Syndrome – remission stage V (end state of Klüver-Bucy-Phase)

Pat. H. P., 36a

- Traumatic apallic syndrome
- Cerebrale MRI: frontal lobe lesions



Traumatic apallic syndrome, patient died in full stage

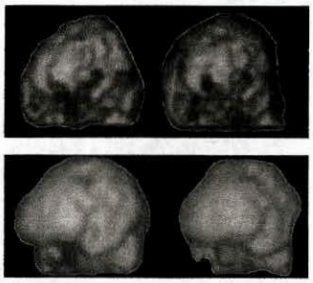


Patient L.G., 32a, death after 9 months after accident
Diffuse white matter lesions, cystic necrosis fronto-temporal, thalamic necrosis, cystic lesions periaqueductal (Heidenhein)

Therapeutic Strategies in apallic syndrome

- Causal therapies in the initial phase (acute midbrain syndrome)
- Special drug treatment (Antispastics, Anticonvulsants, Beta-blockers, Psychostimulants, etc.)
- Stimulation therapies (visual, haptic, accustic, basal stimulation)
- Verticotherapy
- Physiotherapy, ergotherapy, logopedics, cognito therapy
- Therapeutic community, relatives and friends included

HBO-Treatment AS-Remission stage II-III



SPECT:

Marked improvement of perfusion

JN, 21ª, male, traumatic AS, remission stage II-III
HBOT: 64 sessions
1.5 – 1.75 ATA

Additional treatment:
physiotherapy

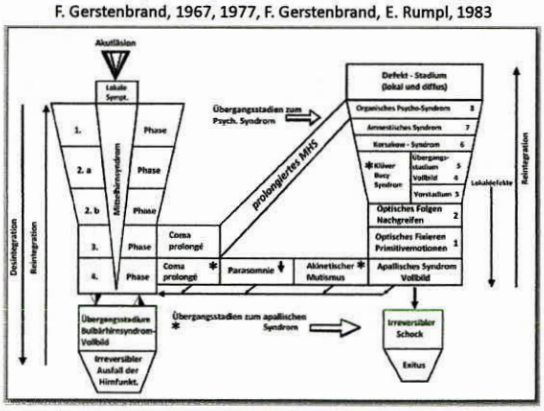
Significant improvement
Defect symptoms:
cerebellar, spastic symptoms, speech disturbances (pseudo-bulbar), cognitive deficits

Prognosis of AS

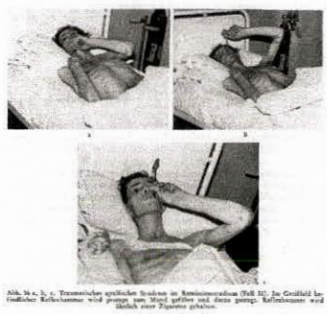
- Can't be made within the first 6 weeks after acute brain damage
- Within the first 6 months no decisions about ongoing of active treatment program possible
- 80% of the patients with an traumatic apallic syndrome develop remission, same post-encephalitic
- 60% of the patients with a hypoxic apallic syndrome show remission, but mostly with severe defects

Traumatic Apallic Syndrome – Course of remission

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



Traumatic Apallic Syndrome, remissions stage IV, Klüver-Bucy-Phase



Patient G.F., 23a
Grasping of objects taking to the mouth, cigarette smoking pattern

Traumatic Apallic Syndrome, remission stage V,
end of Klüver-Bucy-Phase

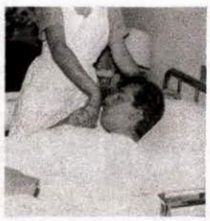
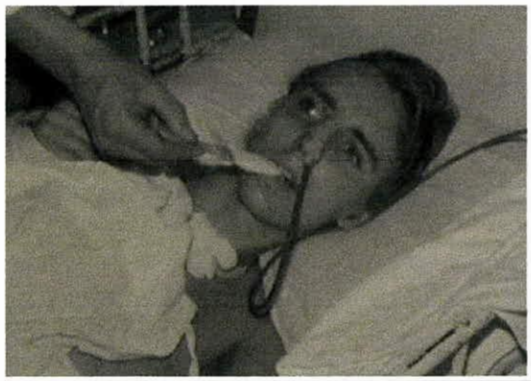


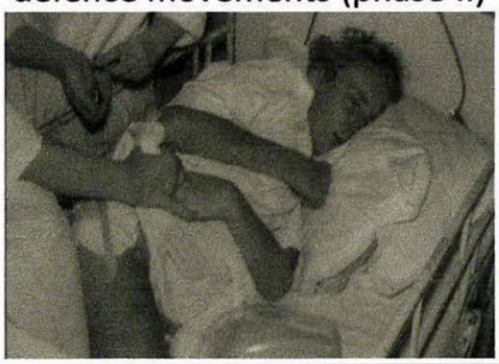
Abb. 37. Traumatisches apallisches Syndrom im Remissionsstadium (Fall 57), Klüver-Bucy-Stadium. Handkuß-Schablone.

Patient A.S., 20a
Handkiss-pattern

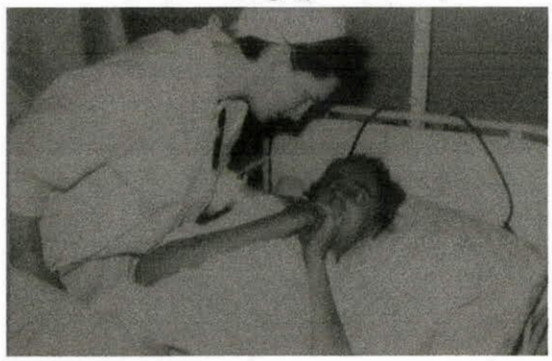
Traumatic apallic syndrome
Full stage, (Peter L., 20 years old)



Early remission stage, initial
defence movements (phase II)



Late remission stage, contact with
surrounding (phase VI)



Traumatic Apallic Syndrome
Full recovery (20 months after accident)

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ROMANIA

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2007

posterior part of the putamen in RT patients and seem to be independent of the D2-receptor status. In conclusion, the presented data corroborate the hypothesis that RT represents a phenotype of PD.

A single examination consisted of two fMRI measurements, when the right and the left hand palm were vibrated. The whole study was performed in six healthy volunteers. For data analysis we used

On **Wednesday 21 March** will take place the **Neurotrauma Session,** under the coordination of Prof. Dr. Pieter Vos from Holland (President of the **EFNS Scientific Panel Neurotraumatology.**

Following speakers are registered to give a lecture within this session - in alphabetical order:

Alekseenko Yuri (Belorussia): Guidelines for early management
Gerstenbrand Franz (Austria): Traumatic Brain Injury, biomechanic aspects and classification
Muresanu Dafin (Romania): Neuroprotection and neuroplasticity in traumatic brain and spinal cord injury
Opara Jozef (Poland): Post neurotrauma Cognitive and motor Rehabilitation
Vos Pieter (Holland): Biomarkers in mild and severe traumatic brain injury
Klaus von Wild (Germany): Prediction of Outcome in traumatic brain injury

Members of the **International Committee and Coordinators** are in alphabetical order:

- **Bajenaru Ovidiu**
- **Korczyn Amos**
- **Muresanu Dafin**
- **Vecsei Laszlo**
- **Vos Pieter.**

Members of the **Local Committee** are in alphabetical order:

- **Marginean Ioan**
- **Panea Cristina**
- **Popescu Bogdan**
- **Popescu Dinu Cristian**
- **Tiu Cristina.**

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