Traumatic Brain Injury Classification

based on biomechanical and neuro-pathological analysis

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Biomechanics of TBI

- Two physical factors are important:
  speed *v*
  acceleration *b*

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

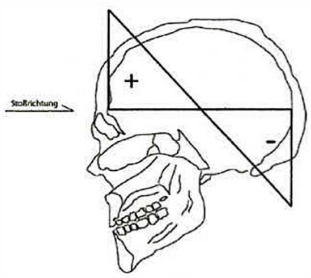
where *s* is the deceleration distance

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)

Biomechanics, physical analysis

Sellier, Unterharnscheidt, 1963



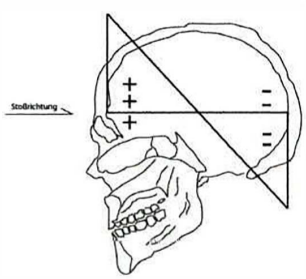
- Positive pressure at the impact pole
- Negative pressure at the counter pole

Different types of TBI

- Closed cerebral trauma
  sometimes combined with fracture of skull
- Open brain trauma by a penetrating object (bullet, etc.)

Biomechanics (impact trauma)

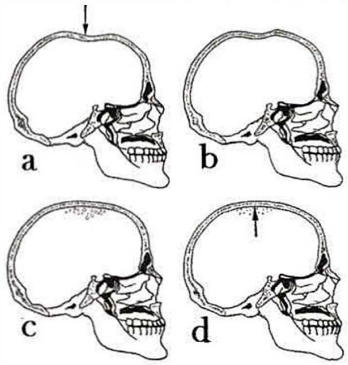
after Sellier and Unterharnscheidt, 1963



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

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

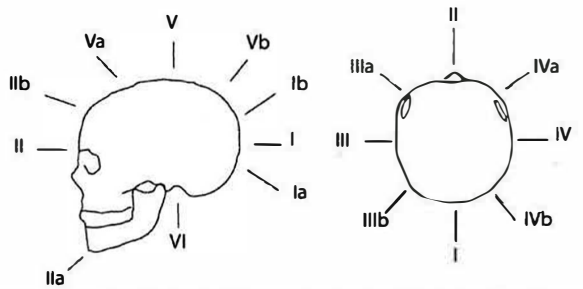
Due to snapping back of the elastic skull bone negative pressure emerges gas bubbles, cortical lesions (d)

Different Types of Brain Trauma
Classification by biomechanical analysis

- Linear brain trauma
  - Linear outer brain trauma
  - Linear inner brain trauma
    - Upper I. BT
    - Lower I.B.T
- Rotational brain trauma

Scheme of traumatic impact in closed skull trauma producing brain lesions

Documentation after Spatz,
modified to Innsbruck impact scheme



Biomechanics
acceleration, deceleration impact

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

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

Traumatic brain lesions

Brain tissue damage depends on

- Form of the impact (blunt, open)
- Direction of the impact
- Location of impact
- Intensity of the force

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)

Patterns of cerebral trauma II

Acceleration – Deceleration trauma

- Rotational brain trauma – lesions:

1. Laceration of brain tissue (capsula int., basal ganglia)
2. Intracerebral haemorrhage (thalamus, hypothalamus)
3. Extracerebral haematoma
   - subdural haematoma – acute, chronic
   - epidural haematoma)

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

- Coup lesions, countre-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

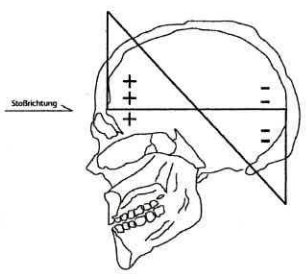
Type of Traumatic Brain Damage I

- Primary lesions, immediately by impact, mostly irreversible
  - Outer brain trauma
  - Inner brain trauma
  - 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

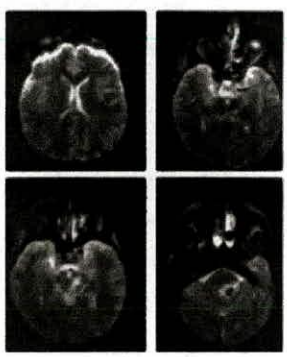
Type of Traumatic Brain Damage II

- Secondary lesions of brain tissue
  1) Consequences of primary lesions, Penumbra
     - Local, regional lesions
  2) Non-cerebral disorders, hypoxia, hypoxemia, circulatory disturbances
     - local, regional, diffuse lesions
  3) Tentorial herniation
     a) local pressure of the 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)

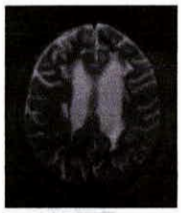
Linear outer brain trauma

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



Linear outer brain injury

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



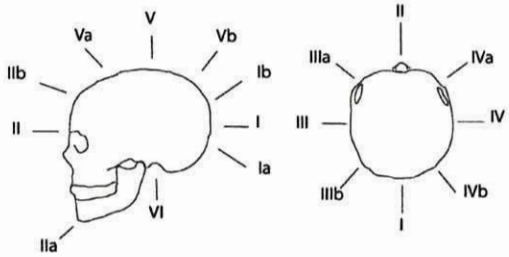
Etiology of brain tissue damage after closed skull trauma – impact scheme

Brain tissue damage depends on

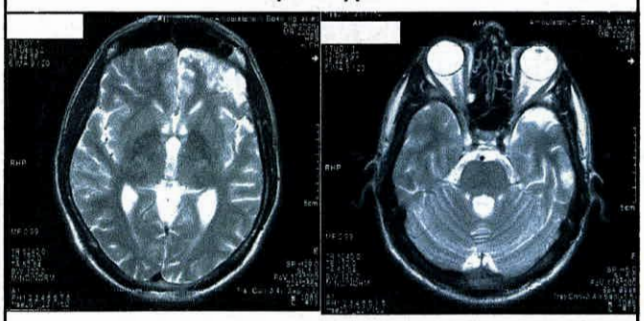
- Direction and form of impact
- Location of impact
- Intensity of the force

Documentation after Spatz, Innsbruck modified

Multiple impacts



Linear outer brain trauma, impact type I



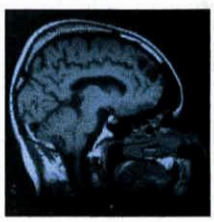
Severe lesions frontal, temporal, minor lesion cerebellar

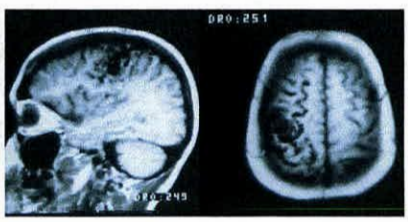
Linear Inner Brain Trauma

Primary Lesions

- **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)

Different types of linear outer brain trauma

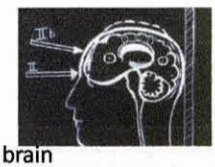


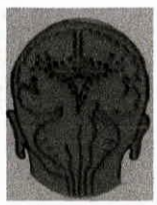


Impact type I Impact Type IV

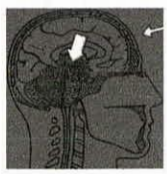
Linear Inner Brain Trauma

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





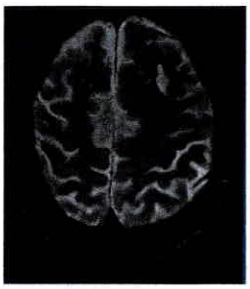
b) Linear inner lower brain trauma (Lindenberg) lesions brain stem, surrounding brain region Type V, Va



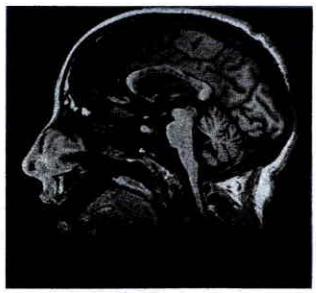
Linear Inner Upper Brain Trauma (GRCEVIC) Type IIb, Ia (II)

- 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 Type Ib

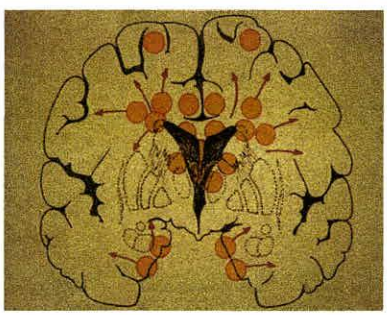


Parasagittal lesion, butterfly type



Lesion corpus callosum, lesions butterfly type

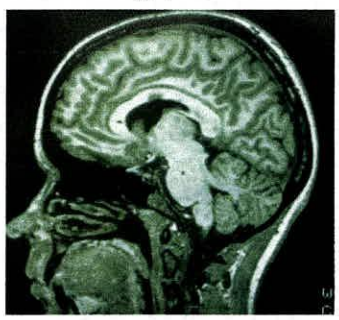
Linear Inner Upper Brain Trauma Schematic drawing (N. Grcevic)



Impact type IIb, Ia, (II)

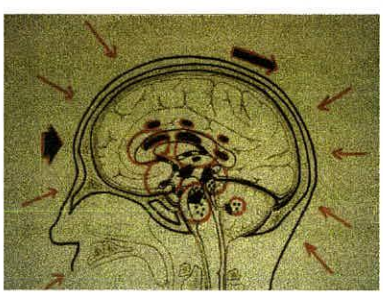
Main lesions, periventricular

Linear Inner Upper Brain Trauma, Impact Type IIb



Local lesion corpus callosum

Linear Inner Upper Brain Trauma Combination with rotational trauma Schematic drawing (N. Grcevic)

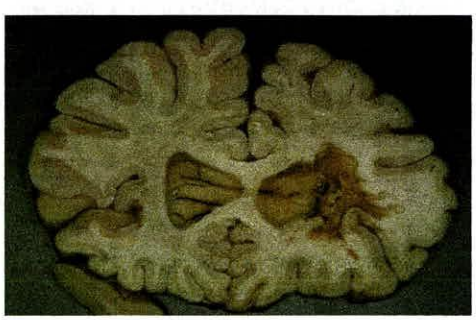


Impact type II, IIa, often with rotational component

Lesions, periventricular, upper brain stem

Boxing impact frontal region

Linear Inner Upper Brain Trauma Type Ib

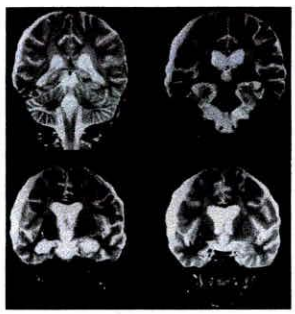


Frontal white matter, periventricular damage

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

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

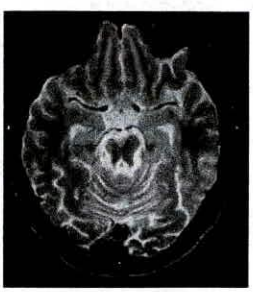
Linear Inner Lower Brain Trauma Combination type Va, IVa



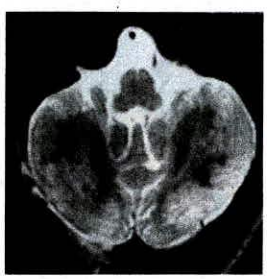
Lesions hippocampal, parahippocampal

Hygrom fronto-parietal left side

Linear Inner Lower Brain Trauma Type Va, Primary lesions



Mesencephalon

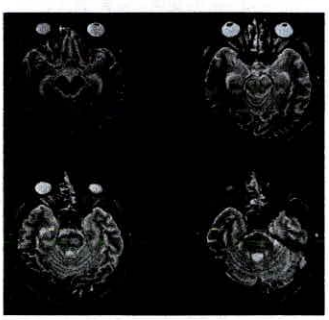


Cerebellum

Linear Inner Lower Brain Trauma, Type Va, Primary lesions

Gliotic lesions with haemosiderin deposition, lower midbrain, pons

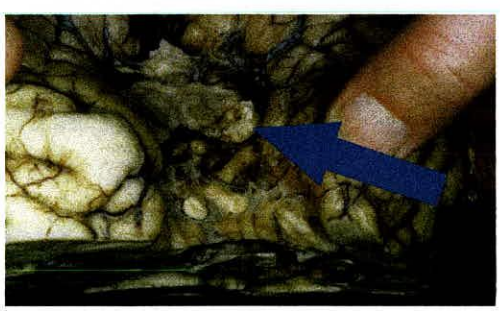
Linear Inner Lower Brain Trauma Type Va, Combination with tentorial herniation



Primary lesion pons,medulla oblongata, (upper part)

Secondary lesion, by tentorial herniation lower midbrain

Linear Inner Lower Brain Trauma Combination with uncal tentorial herniation



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

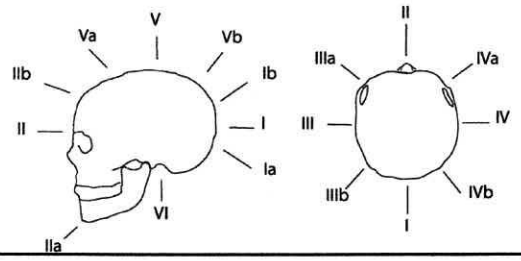
Etiology of brain tissue damage after closed skull trauma – impact scheme

Brain tissue damage depends on

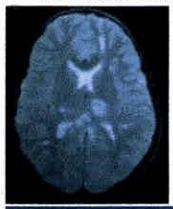
- Direction, form of impact
- Location of impact
- Intensity of the force

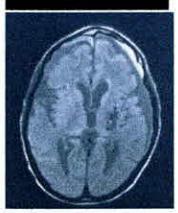
Multiple impacts possible

Documentation after Spatz, Innsbruck modified



Rotational Trauma, H.R., 28a



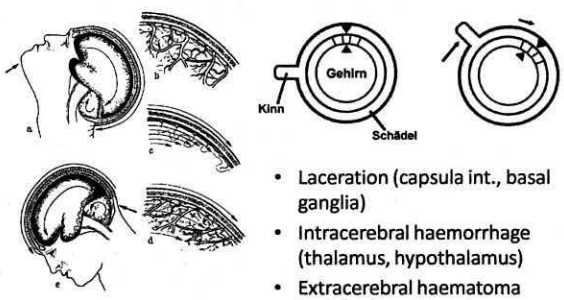


Thalamic haematomas both sides
Laceration internal capsle left

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

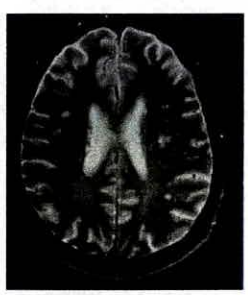
Control MRI after 6 months

Rotational trauma – Scheme Pudenz-Shelden

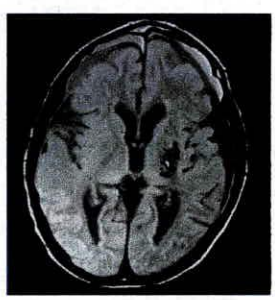


- Laceration (capsula int., basal ganglia)
- Intracerebral haemorrhage (thalamus, hypothalamus)
- Extracerebral haematoma (subdural, epidural)

Rotational Brain Trauma Type IIb



White matter lesions, small haematoma



Lesions: basalganglia, capsula interna

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

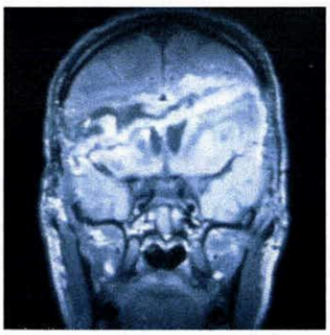
- Intracerebral laceration (basal ganglia, capsula interna)
- Intracerebral hematoma (thalamus, hypothalamus)
- Extracerebral hematoma (subdural, epidural)

Open Brain Trauma

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

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

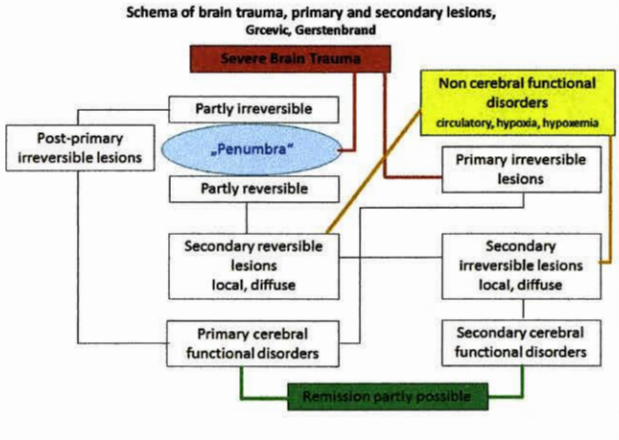
Open Brain Trauma



Bullett injury, suicide, brain death

Classification of brain trauma

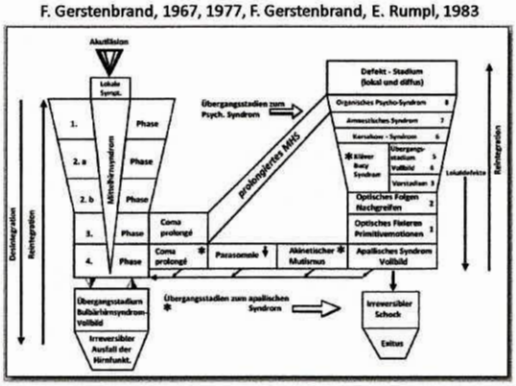
- Mild traumatic brain injury
  (brain commotion, Commotio Cerebri, Hirnerschütterung)
  Glasgow Coma Scale (GCS) = 13 – 15
- Moderate traumatic brain injury
  (brain contusion, Contusio Cerebri – mild degree)
  GCS = 9 – 12
- Severe traumatic brain injury
  (brain contusion, Contusio Cerebri – severe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



Secondary acute midbrain syndrome,

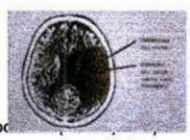
further course

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



Different forms of traumatic lesions

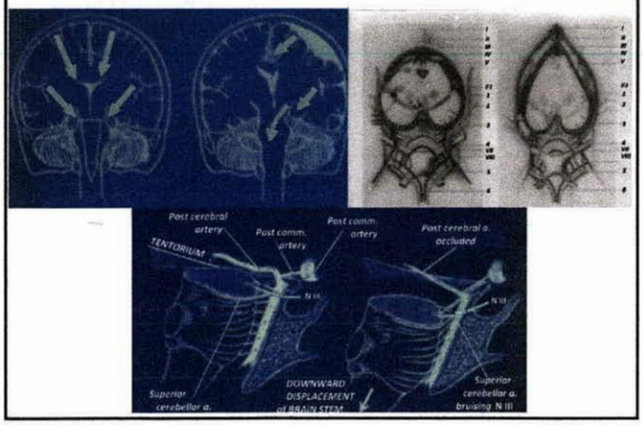
- Primary lesions (irreversible)
- Secondary lesions (therapeutic battle field)



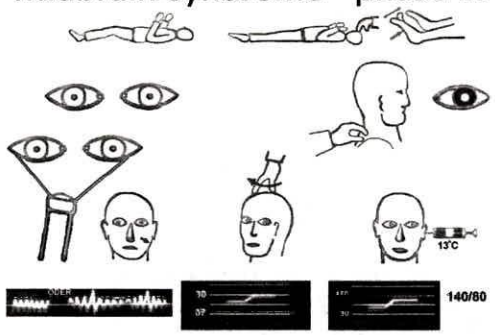
Penumbra, postedemic, posthypoxic, posthyp

- 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

Supratentorial volume increase



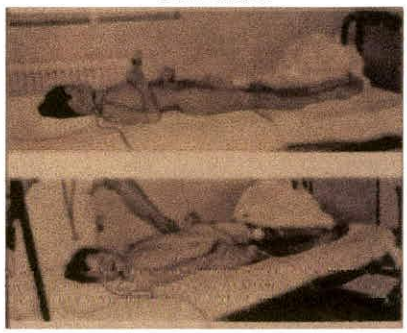
Midbrain Syndrome - phase III



Phase III, Stretch position, disinhibition of vegetative system

Acute secondary midbrain syndrome

Brain edema



Phase III, IV

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

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	NORMAL	BEGINNENDE DIVERGENZ	DIVERGENZ	DIVERGENZ	DIVERGENZ FIXIERT	DIVERGENZ FIXIERT
		PENDELND	SCHWIMMEND	DYSKUNAUGIERT	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

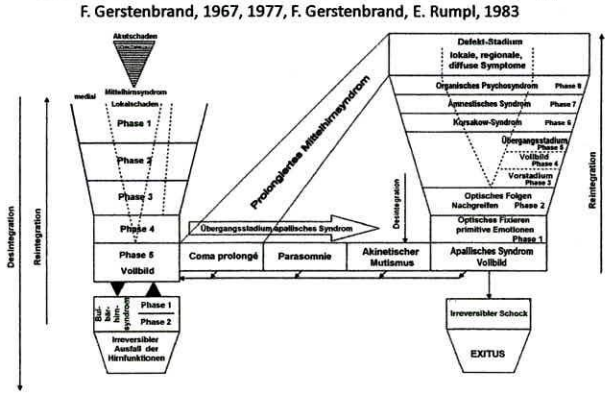
Acute secondary midbrain syndrome

Brain edema

Phase III, IV

Severe Brain Trauma (primary, secondary)

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



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 syndrome, brain death

Preclinical Management

- Care for vital function
  Respiration (orotracheal intubation, if necessary)
  Stabilization of Blood circulation (infusion)
- Documentation of the impact (Spatz – Innsbruck Scheme)
- Registration of secondary injuries

Acute Traumatic Midbrain Syndrome
Secondary etiology

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

Management by the admitting hospital

- Control of vital functions
  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 control
- Decision to transfer the patient in the ICU
- Begin of rehabilitation program

Management of Severest Brain Trauma
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

First measurements in the ICU

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

Treatment of brain edema

- Osmotic therapy
- Diuretic therapy
- Barbiturate
- Hyperventilation

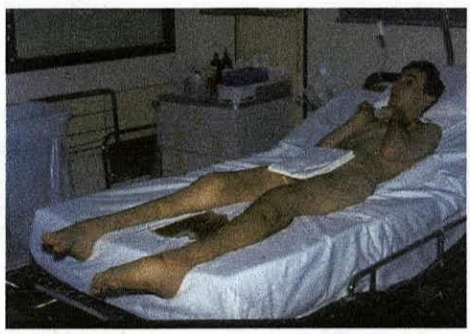
Apallic Syndrome (AS)
after acute severe brain trauma

- 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)

Special methods in treatment of brain edema

- Hypothermia (mild, 32° - 34°)
- Craniotomy (both sides) in cases with progression

Transition stage to apallic syndrome

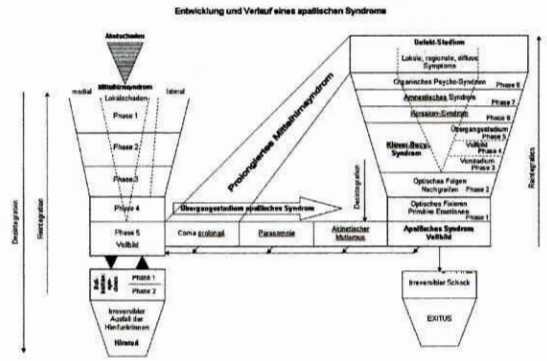


R. S., 26ª

Severe traumatic brain injury

Rest symptoms of an acute midbrain syndrome phase IV

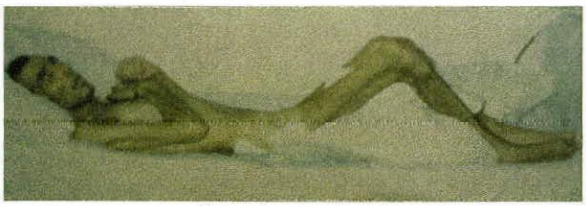
Severest Brain Trauma, further course
F. Gerstenbrand, 1967, 1977, F. Gerstenbrand, E. Rumpl, 1983



Symptoms of AS

- Coma vigile
- No recognition of the surrounding
- No contact to the surrounding
- No reaction to external stimuli
- Sleep-wake-rhythm fatigue regulated
- Optomotoric disturbances
- Flexed-stretched position of the extremities and trunk
- Rigido-spasticity
- Primitive motor patterns (oral, grasping, etc.)
- Dysregulation of the vegetative system

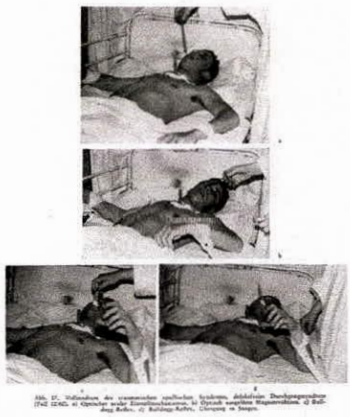
Apallic syndrome, pat. G.B., 36a
traumatic brain injury, 1975



No modern treatment

Irreversible tertiary lesions, complications

Exitus after 14 months



Pat. G.N., 39a

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

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

Modern treatment program in special center for apallic syndrome patients

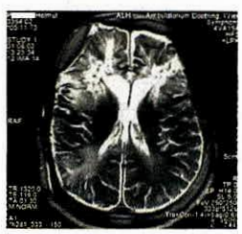
No tertiary lesions, minimal complications

Remission after 5 months to minimal defect state

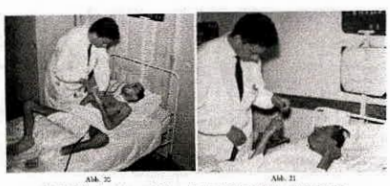
Traumatic Apallic Syndrome – remission stage V
(end state of Klüver-Bucy-Phase)

Pat. H. P., 36a

Traumatic apallic syndrome
Cerebrale MRI: frontal lobe lesions



Traumatic Apallic syndrome
Full stage, primitive motor patterns



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

Traumatic Apallic Syndrome, patient died in full stage

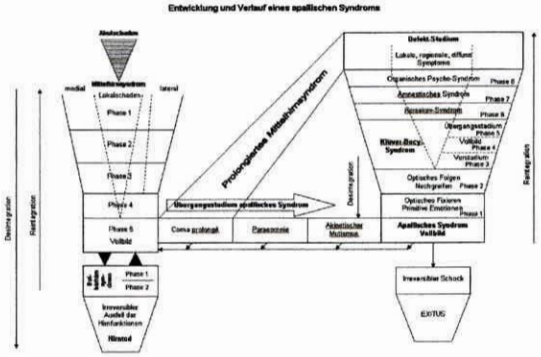
Patient L.G., 32a, death after 9 months after accident
Diffuse white matter lesions, cystic necrosis fronto-temporal, thalamic necrosis, cystic lesions periaqueductal (Heidenhein)

Therapeutic Strategies in Apallic Syndrome

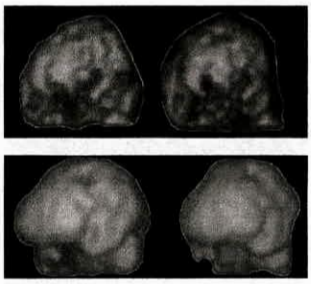
- Causal therapies in the initial phase (acute midbrain syndrome)
- Special drug treatment (antispastics, Anticonvulsants, ß-blockers, psychostimulants, etc.)
- Stimulation therapies (visual, haptic, acustic, basal stimulation)
- Verticotherapy
- Physiotherapy, ergotherapy, logopedia, cognitotherapy
- Therapeutic community, relatives and friends included

Traumatic Apallic Syndrome – Course of remission

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



HBO-Treatment
AS-Remission stage II-III



SPECT:

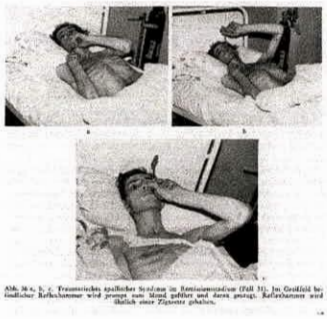
Marked improvement of perfusion

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

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



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

Prognosis of AS

- Can't be made in 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 develop remission, but mostly with severe defects

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

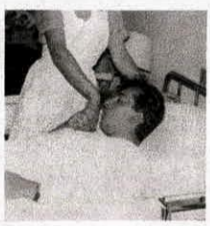
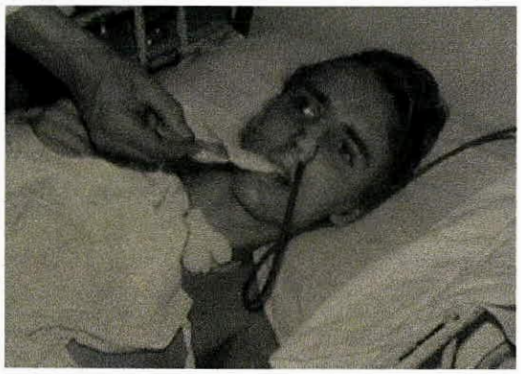


Abb. 37. Traumatisches apallisches Syndrom im Remissionsstadium (Fall 87), Klüver-Bucy-Stadium. Handkuß-Schablone.

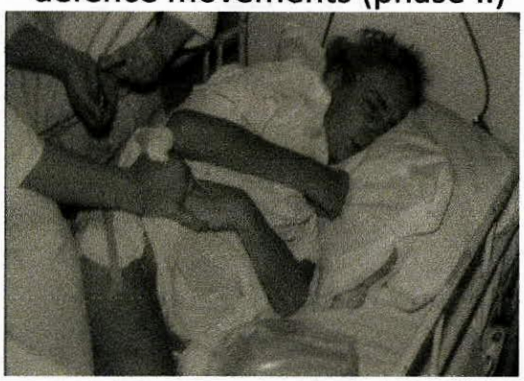
Patient A.S., 20a
Handkiss-pattern

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



Traumatic Apallic Syndrome
Full recovery (20 months after accident)

Early remission stage, initial defence movements (phase II)



Late remission stage, contact with surrounding (phase VI)