Karl Landsteiner Institute for Neurorehabilitation and Space Neurology

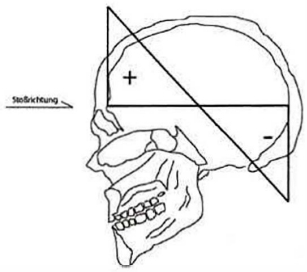
Traumatic Brain Injury a demand for Neurology

F. Gerstenbrand [1), 2)], Ch.Kurzmann [1),]

1) Karl Landsteiner-Institute for Neurorehabilitation and Space Neurology, Vienna, Austria
2) Department of Neurology, Medical University Innsbruck, Austria

Neurological Teaching Course
World Federation for Neurology
February 1, 2013,
No.2 Military Hospital, Yangon, Myanmar

Biomechanics, physical analysis
Sellier, Unterharnscheidt, 1963

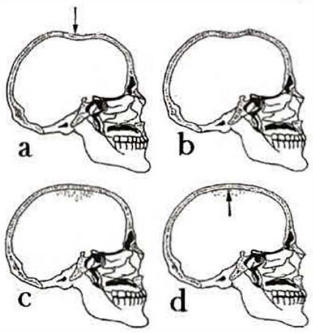


- Positive pressure at the impact pole
- Negative pressure at the counter pole

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, 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

Different types of TBI

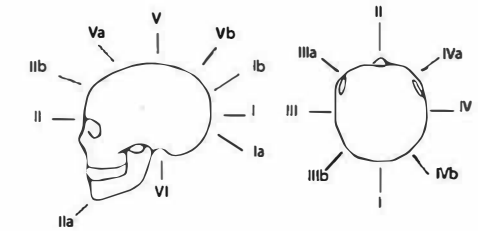
- Closed cerebral trauma sometimes combined with fracture of skull
- Open brain trauma by a penetrating object (bullet, etc.)

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

Documentation after Spatz, Innsbruck modified

Multiple impacts possible



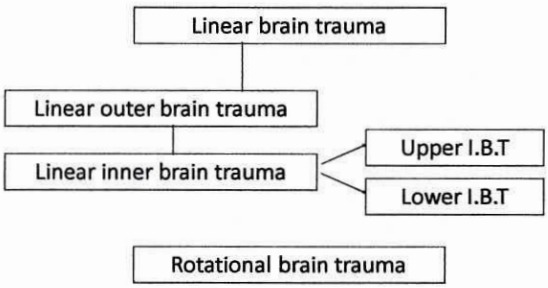
Patterns of cerebral trauma
Acceleration - Deceleration

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

Traumatic Brain Damage II

- Secondary lesions of brain tissue
  1) Umbra/Penumbra, primary impact
     - regional lesions
  2) Non-cerebral disorders caused by hypoxia, hypoxemia, circulatory disturbances
     - local, regional, diffuse lesions
  3) Tentorial herniation
     a) local damage due to tentorial edge
        - local lesions (upper brain stem, medial temporal lobe)
        - 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)

Different Types of Brain Trauma
Classification by biomechanical analysis



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

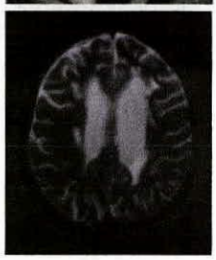
- Coup lesions, contre-coup lesions
  – Cortical, sub-cortical, meningeal damage, funnel-shaped
    - Type I severe lesions fronto-temporal
      Contre-coup negative pressure
    - Type II minor lesions frontal
      force absorption by facial skeleton
    - Type III, IV mostly combined with rotational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

Traumatic Brain Damage I

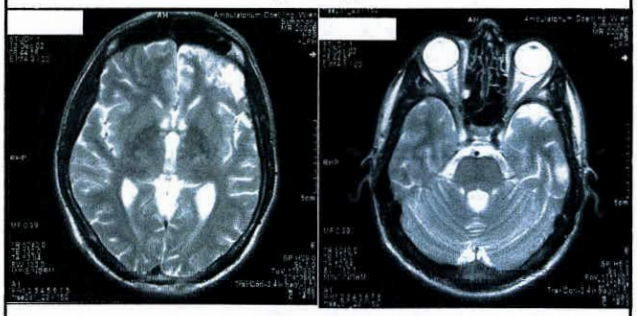
- Primary lesions, immediately by impact, mostly irreversible
  – Outer brain trauma
  – Inner brain trauma
  – Rotational brain trauma

Linear outer brain injury

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



Linear outer brain trauma, impact type I



Severe lesions frontal, temporal, minor lesion cerebellar

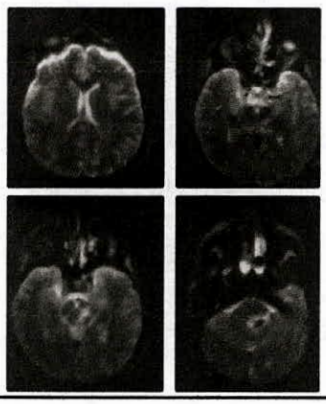
Linear Inner Brain Trauma

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)

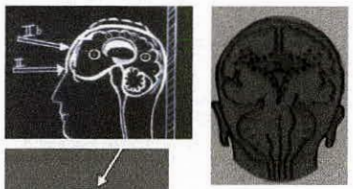
Linear outer brain trauma

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

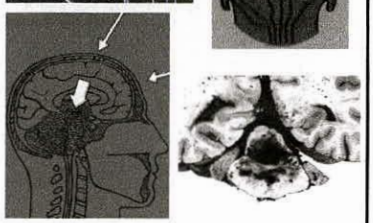


Linear Inner Brain Trauma

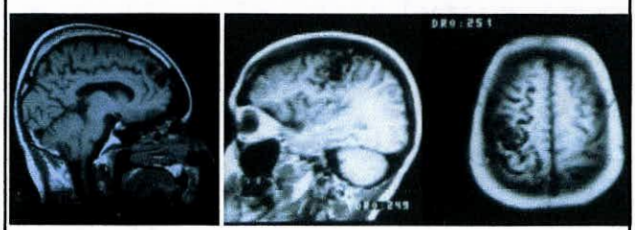
a) Linear inner upper brain trauma (Grcevic) butterfly lesions
Type IIb, Ia (II)



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



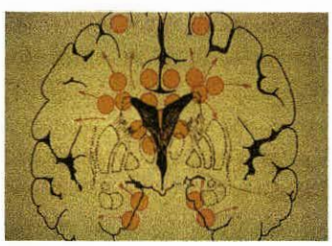
Different Types of Linear Outer Brain Trauma



Impact type I

Impact type IV

Linear Inner Upper Brain Trauma Schema, originally drawn by N. Grcevic

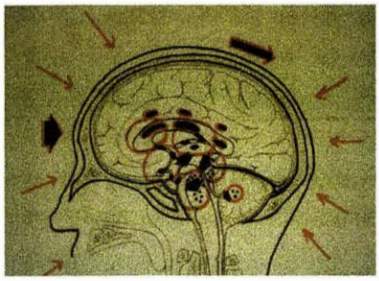


Impact type IIb, Ia, (II)

Main lesions, periventricular

Partly lesions hippocampal area, frontal

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

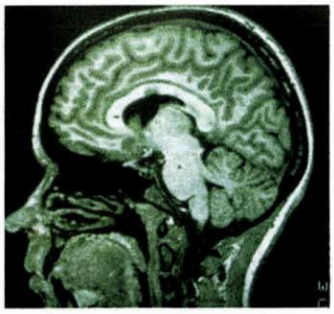


Impact type II, IIa, often with rotational component

Lesions, periventricular, upper brain stem

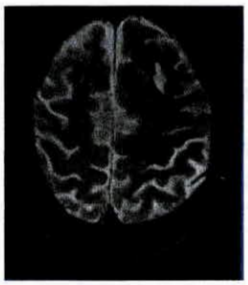
Boxing impact frontal region

Linear Inner Upper Brain Trauma, Impact Type IIb

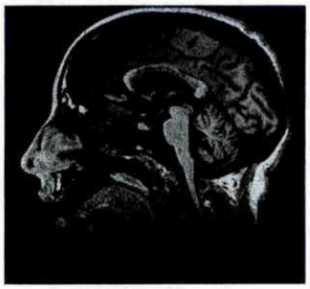


Lesions corpus callosum

Linear Inner Upper Brain Trauma Type Ib



Paraventricular lesion, butterfly type



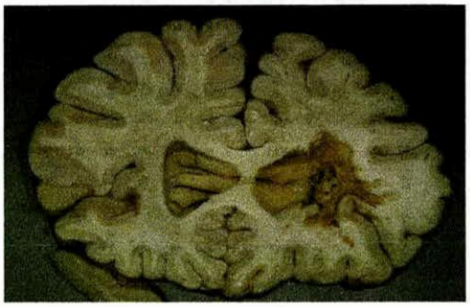
Lesion corpus callosum

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

- Direct lesions
  - upper brain stem
  - surrounding brain region
    - medial temporal lobe
    - cerebellum
- Indirect lesions: tentorial contusion
  - medial temporal lobe
  - Tentorial edge impact

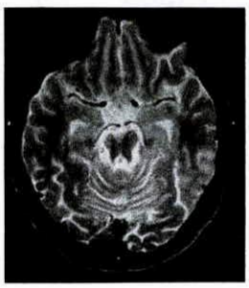
    a) local pressure, upper brain stem (local lesions)
    b) compression of arteries, veines (regional lesions)

Linear Inner Upper Brain Trauma Type Ib

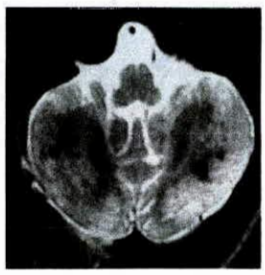


Frontal white matter, periventricular damage

Linear Inner Lower Brain Trauma Type Va, Primary lesions

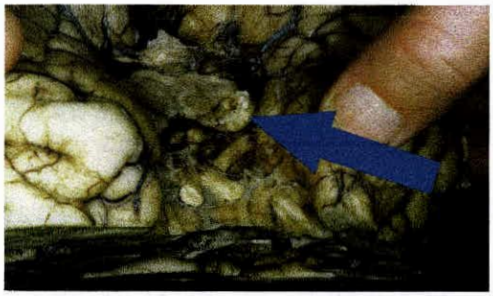


Mesencephalon, temporal lobe



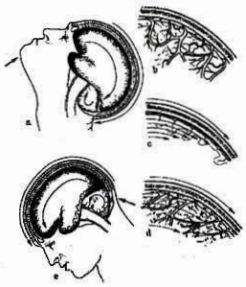
Cerebellum, upper brain stem

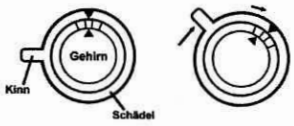
Linear Inner Lower Brain Trauma (Type Va)
Combination with Rotational Trauma (IVa)
Uncal Tentorial Herniation



Direct lesion in the upper midbrain, idirect lesion after uncal herniation (arrow)

Rotational trauma – Scheme
Pudenz-Shelden





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

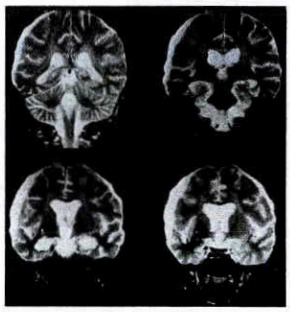
Linear Inner Lower Brain Trauma, Type Va, Primary lesions

Gliotic lesions with haemosiderin deposition, lower midbrain, pons

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)

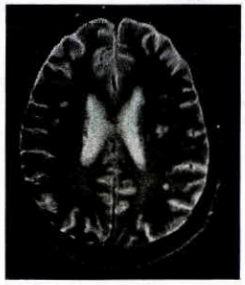
Linear Inner Lower Brain Trauma (Type Va),
Combination Rotational Brain Trauma
(Type IVa)



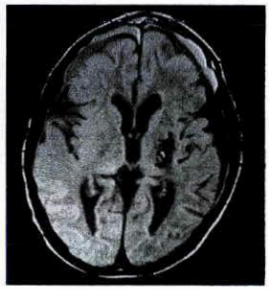
Lesions hippocampal, parahippocampal midbrain

Hygroma fronto-parietal right side, minimal left side

Rotational Brain Trauma
Type IIb

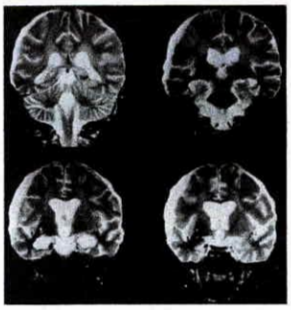


White matter lesions, small haematoma



Lesions:
basalganglia, capsula interna

Linear Inner Lower Brain Trauma (Type Va), Combination Rotational Brain Trauma (Type IVa)

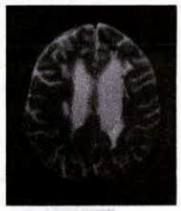


Lesions hippocampal, parahippocampal midbrain

Hygroma fronto-parietal right side, minimal left side

Linear outer brain injury

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



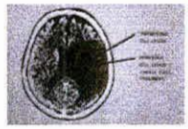
Open Brain Trauma

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

Direct brain lesion with consecutive brain edema and hematoms

Different forms of traumatic lesions

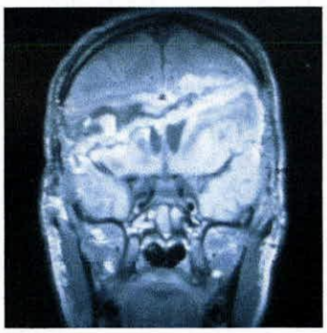
- Primary lesions (irreversible)
- Secondary lesions (therapeutic battle field)



Penumbra, postedemic, posthypoxic, posthypoxemic (diffuse/local)

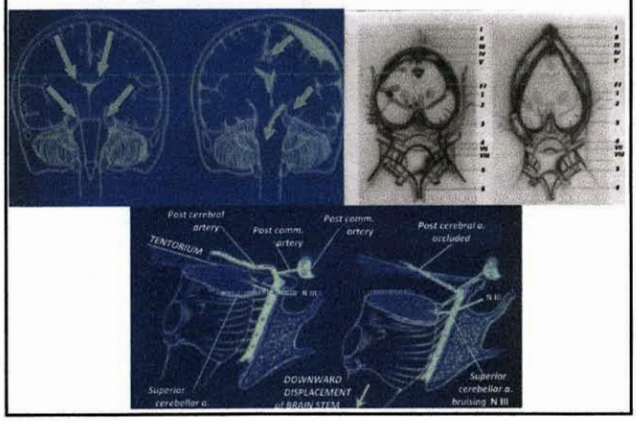
- Tertiary lesions (malnutrition, malabsorption, avitaminosis, bed rest syndrome, etc.)
  Encephalopathy, myelopathy, pontine myelinolyse, polyneuropathy
- Quartary lesions
  hydrocephalus occlusus, meningoencephalitis, brain abscess
- Complications
  joint contraction, periarticular ossification, decubitus, pressure lesion of peripheral nerves

Open Brain Trauma

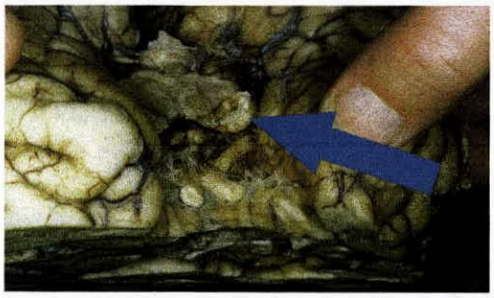


Bullett injury, suicide, brain death

Supratentorial volume increase



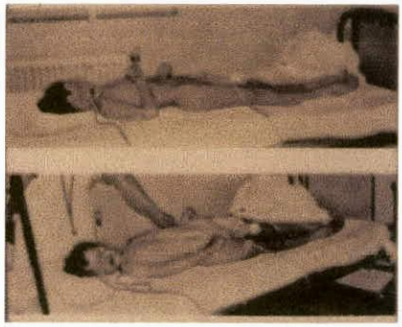
Linear Inner Lower Brain Trauma (Type Va)
Combination with Rotational Trauma (IVa)
Uncal Tentorial Herniation



Direct lesion in the upper midbrain, idirect lesion after uncal herniation (arrow)

Acute secondary midbrain syndrome

Brain edema

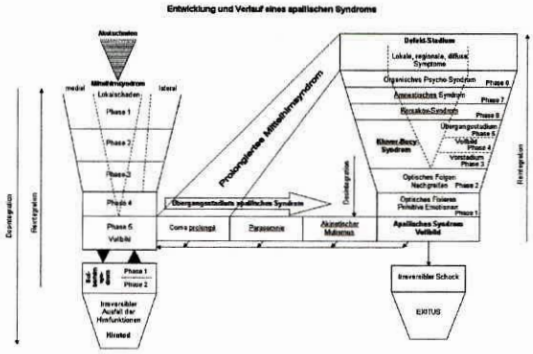


Phase III, IV

Severe Brain Trauma

further course

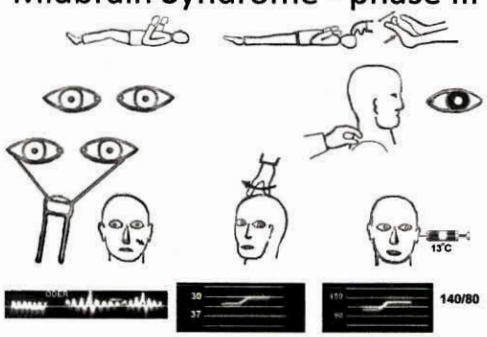
F. Gerstenbrand, 1967, 1977, F. Gerstenbrand, E. Rumpl, 1983



Midbrain syndrome phase IV

- Coma
- Missing blink reflex and ocular movements
- Divergent position of bulbi
- Pupils reduced reaction to light
- Oculocephalic reflex disturbed (dull head phen.)
- Vestibuloocular reflex dissociated reaction
- Stretch position of the extremities, stretch synderg.
- Increased muscle tone, pyramidal signs, hyperrefl.
- Respiration – machine like rythmus
- Hyperthermia, tachycardia, increased blood pressure

Midbrain Syndrome - phase III



Phase III, Stretch position, disinhibition of vegetative system

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-SYNER-GISMEN	REST-STRECK-SYNERGISM.	FEHLEND
OPTOMOTORIK	-STELLUNG BULBUS -BEWEGUNG	NORMAL	NORMAL	BEGINNENDE DIVERGENZ	DIVERGENZ	DIVERGENZ	DIVERGENZ FIXIERT	DIVERGENZ FIXIERT
		PENDELND	SCHWIMMEND	DYSKINUGIERT	FEHLEND	FEHLEND	FEHLEND	FEHLEND
	PUPILLENWEITE	● ●	● ●	● ●	● ●	● ●	● ●	● ●
	LICHTREAKTION	● ●	● ●	● ●	● ●	● ●	● ●	● ●
KÖRPER-MOTORIK	KÖRPERHALTUNG							
	SPONTAN-MOTORIK	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 VEGETATIV	ATMUNG							——
	PULS	LEICHT ERHÖHT	NORMAL	BESCHLEUNIGT	BESCHLEU-NIGT	STARK BESCHLEU-NIGT	BESCHLEU-NIGT	VERLANGSAMT
NICHT OBLIGAT	RR	NORMAL	NORMAL	NORMAL	LEICHT ERHÖHT	ERHÖHT	NORMAL	ERNIEDRIGT
	KÖRPER-TEMPERATUR	NORMAL	NORMAL	LEICHT ERHÖHT	ERHÖHT	STARK ERHÖHT	ERHÖHT	NORMAL ERNIEDRIGT

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

Measurements at ICU

- Stabilisation of vital functions
- Intubation
- Central venous catheter
- Bladder catheter
- Analgosedation (brain stem syndrome)

- Treatment of brain oedema (obligatory)

- Control of cCT
- If possible cMRI
- ICP-control

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

Treatment of brain edema

- Osmotic therapy
- Diuretic therapy
- Barbiturate
- Hyperventilation

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

Management in the admitting Hospital

- Control of vital functions
  Artificial respiration if necessary
  Support of blood circulation (infusion, medication)
- Treatment of brain edema

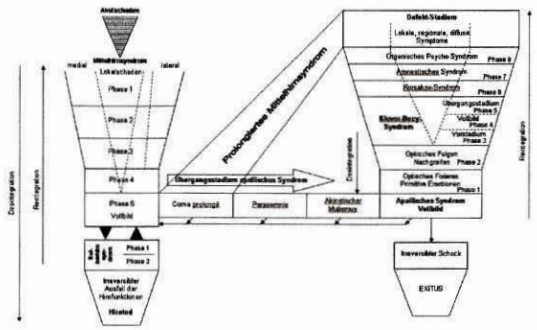
- Neurological status
- Cerebral CT
- X-Ray of cervical spine, skull
- Neurosurgical control
- Decision to transfer the patient to the ICU

- Begin of rehabilitation program

Special methods in treatment of brain edema

- Hypothermia (32° - 34°/mild 35°)
- Craniotomy (both sides)
  in cases with progression

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



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

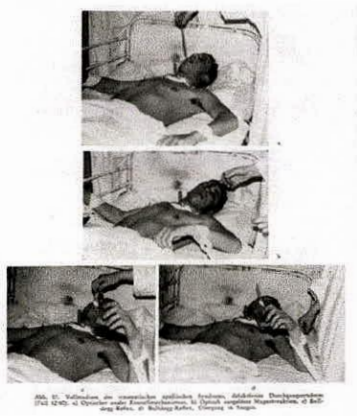
Modern treatment program in special center for apallic syndrome patients

No tertiary lesions, minimal complications

Remission after 5 months to minimal defect state

Apallic Syndrome (AS) / Vegetative State after acute severe brain trauma

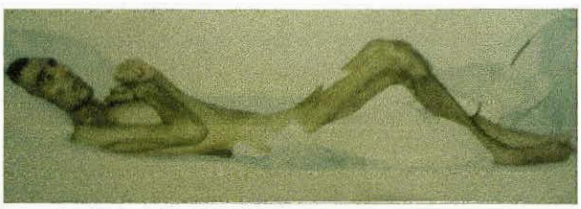
- Initial stage:
  - acute midbrain syndrome (central 5 phases, lateral 2 phases – transmission in phase 4, 5)
  - acute bulbar brain syndrome (2 phases)
- Transition stage to AS (3 phases)
- Full stage of AS
- Remission stage (8 phases)
- Defect stage (multilocular lesions, regional lesions, diffuse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



Pat. G.N., 39a

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

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

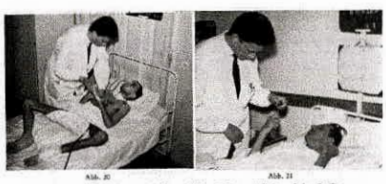


No modern treatment

Irreversible tertiary lesions, complications

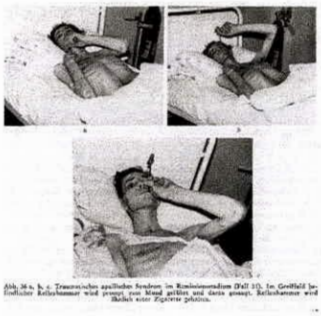
Exitus after 14 months

Traumatic Apallic syndrome
Full stage, primitive motor patterns



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

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



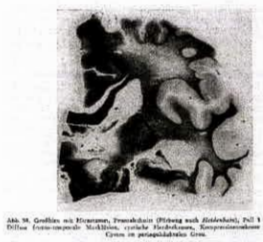
Patient G.F., 23a

Grasping of objects taking to the mouth, cigarette smoking pattern

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, vibrostimulation)
- Verticotherapy
- Physiotherapy, ergotherapy, logopedia, cognitotherapy
- Therapeutic community, relatives and friends included

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)

Prognosis of Traumatic AS

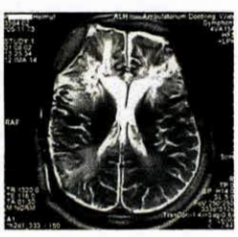
- Within the first 6 weeks in severe and severest cases of traumatic brain injury no prognosis is possible
- Within the first 6 months no decisions about ongoing of active treatment program possible
- 80% of the patients with a traumatic apallic syndrome develop remission
- 25% of the patients with a traumatic apallic syndrome can be re-integrated in normal life
- 60% of the patients with a hypoxic apallic syndrome develop remission, but mostly with severe defects

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

Pat. H. P., 36a

Traumatic apallic syndrome

Cerebrale MRI: frontal lobe lesions, temporal lobe lesions



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, acute bulbar brain syndrome)
  GCS < 5

Severest brain injury
Primary etiology

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

Demands on modern treatment of Traumatic Brain Injury - I

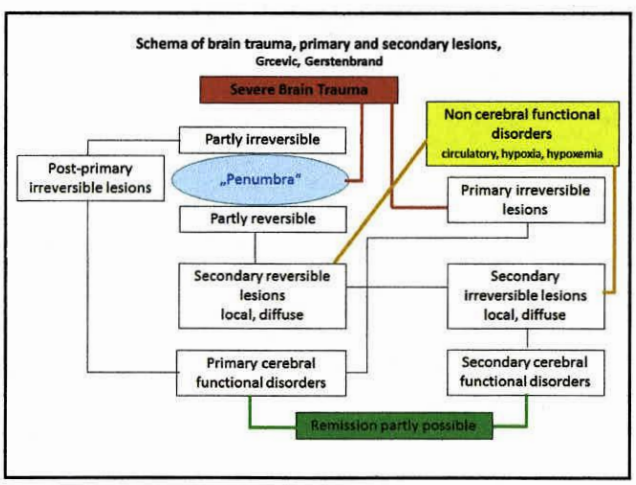
- In initial phase each patient with traumatic brain injury needs an exact neurological diagnosis, mild to severest forms
- Documentation of the impact to the skull (Spatz - Innsbruck Scheme) has to be an obligation
- Severest as well as moderate forms during the initial state have to be treated in ICU (Neurological ICU)

Severest brain injury
Secondary etiology

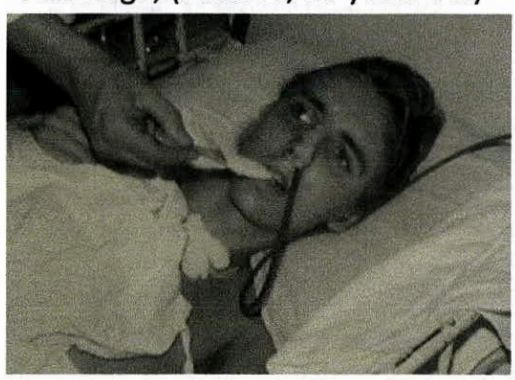
- Due to tentorial herniation (central, uncal)
- Consequence of an increased supratentorial pressure (brain edema; extra-, intracerebral haematomas)
- Symptoms of an acute midbrain syndrome
  Development in 5 phases – central herniation
  Development in 2 phases – uncal herniation – transfer in phase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

Demands on modern treatment of Traumatic Brain Injury - II

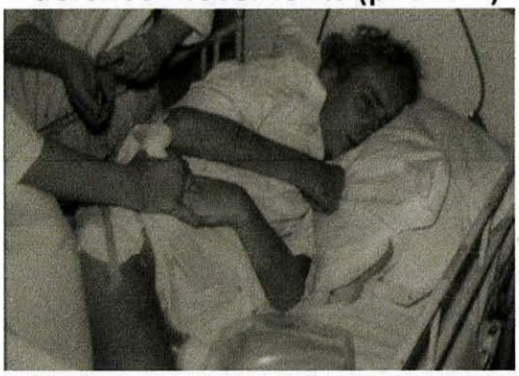
- Early rehabilitation has to be started immediately in all forms of traumatic brain injury (mild to severest)
- In every case an individual neurorehabilitation program is necessary and has to be executed in a special neurorehabilitation center
- The aim of rehabilitation is the reintegration in social life
- Any discussion about preterm ending of treatment and of medical care is ethically not acceptable



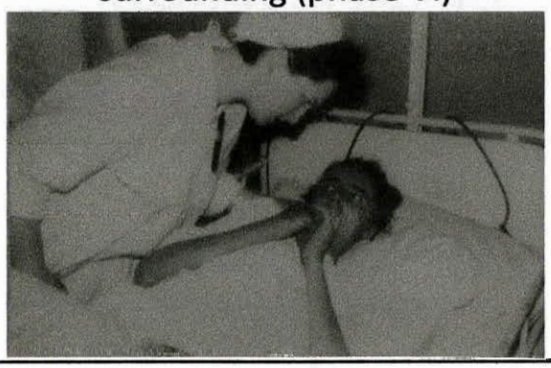
Traumatic apallic syndrome
Full stage, (Peter L., 20 years old)



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)