available that these laboratory estimates of baroreflex sensitivity have both pathophysiologic and clinical relevance, as the sensitivity of the baroreceptor-heart rate reflex seems to be inversely related to mortality rate in myocardial infarction, heart failure and diabetic patients. A deeper insight into the features of daily life baroreflex cardiovascular control has been more recently offered by techniques based on computer analysis of spontaneous blood pressure and heart rate fluctuations, which all share a number of common features. First, these techniques do not require any external intervention on the cardiovascular system thus preventing undesirable interferences with the autonomic function patterns explored. Secondly, they can be used not only to assess BRS in standardized laboratory conditions, but also to investigate the dynamic features of baroreflex modulation of heart rate over time in daily life. Thirdly, arterial baroreflex control of heart rate is explored around the baroreflex 'set point', excluding the portions of the sigmoidal baroreceptor stimulus-response curve approaching threshold and saturation. The information on arterial baroreflex function so obtained appears therefore to be complementary to that provided by the application of conventional laboratory tests. Since the first introduction of methods for spontaneous BRS assessment nearly 20 years ago, a number of papers have supported its pathophysiological and clinical relevance, that however still needs further evaluation.

Quality of life (QoL) as consequence of severe traumatic brain lesions

FW18-1

Quality of life assessment instruments and consequences

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QoLIBRI Despite the dimension of this health plague, there is no specific tool for traumatic brain injury (TBI). E. Neugebauer organised, in October 1999 a systematic literature review and an international expert consensus leading to a research group comprising the representatives of 14 countries and 10 languages, under the aegis of four societies EMN, EBIS, NBIRTT and EBBS and the coordination of JL Truelle. We selected from four tools EBIQ, BICRO-39, SQLP, QoLBI, 6 domains (physical, cognitive, psychological, functional, social and personal) and 56 items with a five points scoring. The validation in process is based on self questionnaires QoLIBRI filled out by TBI and relative, a generic reference SF36, a depression and anxiety scale, the HADS. The examiner fills-in a handicap scale the GOSE (Glasgow Outcome Scale Extended), a co-morbidity and clinical status scale. In each language, five teams will test each 40 patients in 6 months. All the cases will be collected by the methodological centre (N. von Steinbuechel). A new tool, filled out in <20 min, will be reformulated and tested, leading to the final QoLIBRI in 2005. QoLIBRI is intended to represent a metadimension, beyond the handicap, in TBI outcome measures, taking in account the point of view of the patient and family. It assesses the progress of one individual, the efficiency of a therapeutic programme, re-hierarchizes the goals of rehabilitation and is a fast screening of one TBI's troubles and needs.

FW18-2

Early clinical prognostic factors during coma recovery

R. Formisano[1], F. Penta[1,2], U. Bivona[1], M. G. Buzzi[1], P. Ciurli[1], M. Matteis[1], C. Barba[1], C. Della Vedova[1] and V. Vinicola[1]
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Survivors from severe traumatic brain injury (TBI) often suffer from prolonged disturbance of consciousness, such as coma (lasting from few hours to some days), prolonged coma (lasting at least 15 days), minimally conscious state and/or vegetative state. Such conditions may be followed by different clinical outcomes, with recovery process lasting variable periods of time (from few days to several months). Unfortunately, useful studies looking at the possible clinical prognostic factors observed in the early phases of coma recovery are scant. In a previous retrospective study the clinical variable with a significant predictive value on most neuropsychological scores was the interval from head trauma to the recovery of oral feeding. Aim of this study was to confirm the possible prognostic role of some clinical factors emerging during rehabilitation of severe brain injury patients with prolonged coma, also in a prospective study. We enrolled 200 severe brain injury patients, consecutively admitted to the Rehabilitation Hospital Santa Lucia in Rome, from October 2001 to October 2003, as in- or outpatients. Duration of unconsciousness, presence of psychomotor agitation and scores of disability scales such as Disability Rating Scale (DRS) and Levels of Cognitive Functioning (LCF) at entry in rehabilitation were able to predict the outcome at the time of discharge from rehabilitation. A follow up study at 1 year from the discharge time is still in progress.

FW18-3

Assessment of QoL focused in patients after TBI. What and how to measure?

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Abstract not received

FW18-4

The legal basis of treatment

F. Gerstenbrand[1], W. Struhal[2] and H. Baumgartner[3]
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To find better orientation for the course of traumatic brain injury the classification, mild, moderate, severe and severest is demanded. The documentation for the localization of an impact on scull helps in the reconstruction of biomechanical forces in the brain and to categorize the intensity and the local brain lesion. The impact scheme of Spatz (type I–VI) shall be used. An exact diagnose in the acute phase is necessary to decide severity and treatment program. An acute traumatic midbrain syndrome (Gerstenbrand, Luecking), the midbrain – upper pons stage (Plum, Posner) indicates the severest form of a brain trauma. Acute brain stem symptoms can be primer caused by direct damage of the midbrain or secondary due to a tentorial herniation as

sequence of supra-tentorial volume increase (brain edema, intra- or extra-cerebral haematoma). In most of these patients a traumatic apallic syndrome, post-traumatic vegetative state has to be expected. Every patient with severe and severest traumatic brain injury has to be admitted in the intensive care unit. A continuous monitoring and all diagnostic and therapeutic activities have to be initiated immediately, mostly an algo-sedation will be started. After the initial phase of the acute midbrain syndrome lasting during 5–6 days, the transitory phase follows and after 4–6 days the full stage of an apallic syndrome develops (Gerstenbrand). As soon as possible the apallic patient has to be transferred to a special centre, starting with the special programme for apallic patients. Every apallic patient has to be treated, as he would recover completely. In a high percentage (around 35%, Formisano *et al.*) traumatic apallic patients can be resocialized. The main demand is the most early start with a consequent modern neurorehabilitation programme in a highly qualified special centre for apallic patients. The treatment program has to be continued minimally over 6 months with the knowledge, that further recovery is possible. With the diagnostic decision of a chronic apallic syndrome/permanent vegetative state no improvement can be expected. This patient has to be transferred to a nursing home specialized for apallic patients. To prevent negative implication following an interruption of neurorehabilitation measurements a consilium has to be called including the relatives of the patient. The discussion about end of life decision especially in apallic patients in most parts of Europe is not reasonable. Such a decision has to be compared to passive euthanasia, a situation which ethically and legally is not acceptable and can become an act of public prosecutor. Concerning the diagnoses vegetative state the recommendation of the Pro-Life Committee Catholic Bishop Conference in the USA should be seriously discussed. The Bishop Conference has recommended not using the term vegetative state anymore. After the comments of the Pro-Life Committee the term vegetative state would be a degradation of a patient diagnosed as a vegetative state, recalled as vegetable, ending by negative resonance for a human being and offering the danger not to get a proper and efficient treatment programme.

Non-motor manifestations of Parkinson's disease

FW19-1

Non-motor manifestations of Parkinson's disease

A. J. Lees
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Impairment of olfactory discrimination has been recognized as an early and frequent abnormality in Parkinson's disease for 30 years. Abnormalities of smell perception possibly due to amygdala pathology may also occur and a defect in sniffing may also contribute to the hyposmia. In addition to neuronal loss and Lewy body pathology in the anterior olfactory nucleus an increase in dopamine levels in the olfactory bulb has been described. Careful clinical evaluation of olfaction using validated test batteries may be of practical value in differentiating early PD from vascular parkinsonism, 'parkin' disease, atypical tremors and PSP-P. In preparation for the advent of disease-modifying therapies the use of olfaction testing to identify individuals 'at risk' of developing PD is of considerable research interest.

FW19-2

Sleep disturbances in Parkinson's disease

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Having a good night sleep is an essential element of having a good quality of life and it is particularly important to patients with Parkinson's disease (PD) since sleep has a positive, although transient, effect on symptoms (so-called 'sleep effect'). Unfortunately although it is not too uncommon that PD patients complain that they spend nights in misery. It may difficult for some of them to turn or get out of bed and have to urinate frequently. Others may have excessive drooling of saliva, pains in the legs and still others frequent disturbing nightmares. Interest in sleep in PD has been reawaken by recent reports that excessive daytime sleepiness (EDS) can be associated to treatment with dopaminergic drugs. At the same time treatment strategies with long acting dopaminergic drugs or deep brain stimulation that allow for continuous treatment of motor symptoms during both day and night time has sparked interest in the problem with the hope that we can improve nocturnal disability in our patients. In my presentation I will review the major problems that impair sleep quality in PD such as parkinsonism at night and REM behaviour disorder. EDS frequent in PD and in particular in those patients with cognitive deterioration will also be discussed. In these patients REM sleep alterations may be the cause of day time hallucinations. Finally I will review current strategies to treat sleep problems and EDS in PD.

FW19-3

Neuropsychiatric problems in Parkinson's disease

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Parkinson's disease is frequently associated with a variety of neuropsychiatric symptoms encompassing both cognitive and behavioral changes. The prevalence of dementia is reported to be close to 30%, behavioral symptoms are variably reported in 20–80% of patients. Neuropsychiatric symptoms are often a more significant source of distress for patients and families; they significantly impair the quality of life and are often the main reason for nursing home placement. Many patients are found to have cognitive deficits in neuropsychological testing although they are clinically not demented. The prototype of dementia seen in PD is a dysexecutive syndrome. Personality changes, agitation, aggression, mood abnormalities, sleep disturbances, hallucinations and delusions can all occur in PD patients with or without dementia. The first step in the management of behavioral symptoms involves assessment of the underlying cause. Acute confusion due to systemic disorders such as dehyratation or infections, drug-induced confusion or psychosis, social or inter-personal relations should all be considered. Behavioral symptoms may spontaneously abate once exogenous sources are identified and managed. These symptoms, however, may also be due to the underlying disease pathology and may necessitate pharmacological intervention.

Quality of life as a consequence of severe traumatic brain lesions

including vegetative state/apallic syndrome
Medical facts, ethical and legal dilemma

The legal basis of treatment

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8th EFNS Congress
September 4-7, 2004
Paris, France
Special Meeting Neurotraumatology

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

Classification of traumatic brain injury

Closed traumatic 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 – upper brain stem symptoms (acute midbrain syndrome, bulbar brain syndrome)
  GCS < 5

Open traumatic brain injury

Patterns of cerebral trauma

- Acceleration – Deceleration trauma

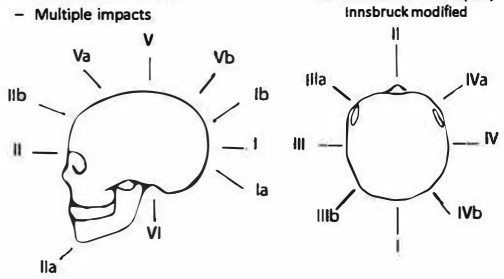
  Linear brain injury
  - Outer brain injury
    1. Coup - local lesions on the impact region
    2. Contre coup - opposite to the impact
  - Inner brain injury
    1. Inner upper brain injury – lesions: corpus callosum, septum pellucidum, fornix, thalamus, hypothalamus, cingulum
    2. Inner lower brain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

- Rotational brain trauma
  1. Laceration (capsula interna, basal ganglia)
  2. Intracerebral hemorrhage (thalamus, hypothalamus)
  3. Extracerebral hematoma (subdural, epidural)

Etiology of brain tissue damage after closed skull trauma

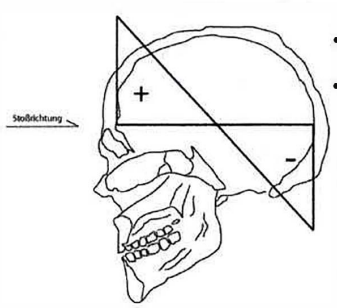
Brain tissue damage depends on

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

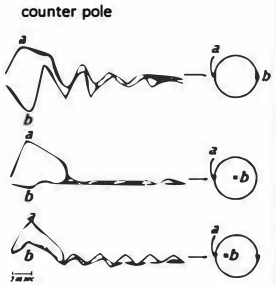


Documentation after Spatz, Innsbruck modified

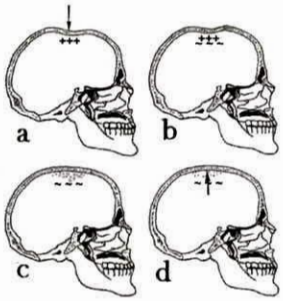
Biomechanics 1



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



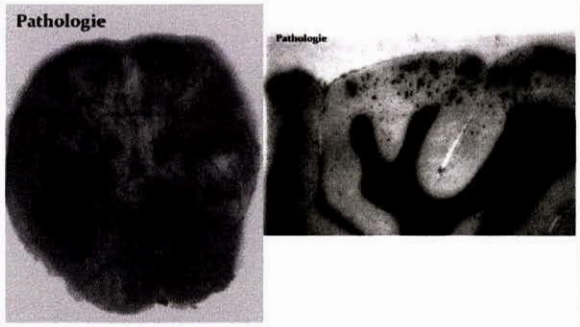
Biomechanics 2



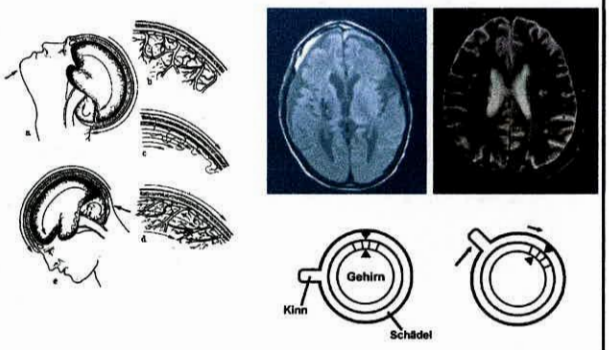
- Damage on the counter pole: Negative pressure causes tissue damage due to gas bubbles (gas, solved in tissue under normal pressure)
- Damage on the impact pole:
  a) Direct damage due to contact of the skull bone leads to lesions on the surface
  b) Due to snapping back of the elastic skull bone negative pressure emerges gas bubbles

Biomechanics 3

Lesions on the surface of the brain



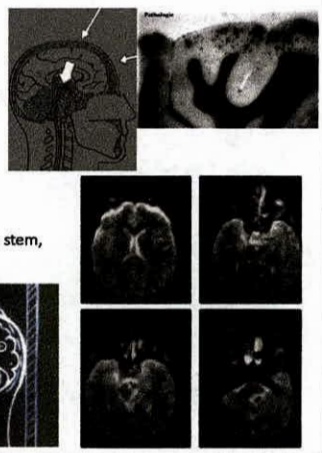
Biomechanics 4
Rotational trauma



Biomechanics 5

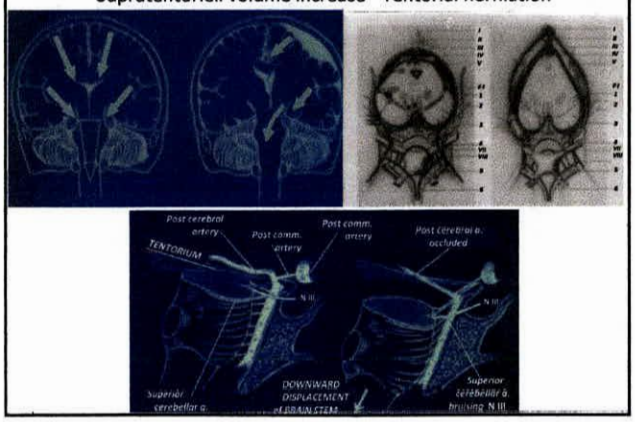
Linear brain injury

- Linear outer brain injury
  Lesions on brain surface depending to direction, intensity and localisation, of the contusion zones
- Linear inner brain injury
  - a) Linear inner upper brain injury (Grcevic) lesions periventricular
  - b) Linear inner lower brain injury (Lindenberg,) lesions upper brain stem, temporal lobe, cerebellum



Biomechanics 6

Supratentoriell volume increase - Tentorial herniation

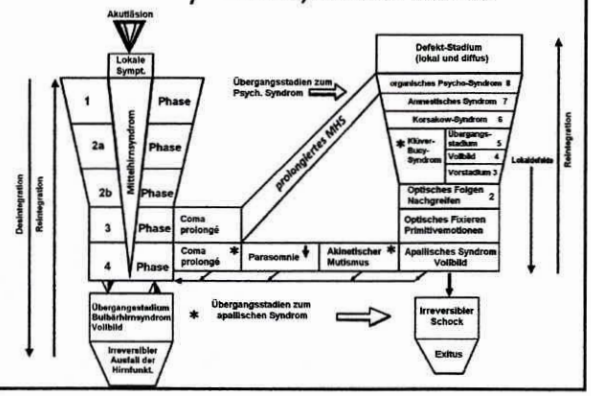


Severest brain injury

- Primary: Direct lesion of the upper brain stem, linear inner lower brain injury (Lindenberg), impact Type V, Va
- Secondary: Tentorial herniation

- Clinical symptoms: Acute midbrain syndrome
- In some cases an acute bulbar brain syndrome will develop
- Bad prognosis, apallic syndrom, brain death

Development of secondary acute midbrain syndrome, further course



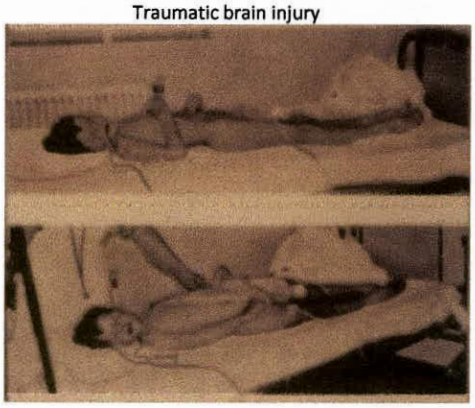
Management of severest traumatic brain injury

4 Phases

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

Acute midbrain syndrome

Traumatic brain injury



Symptoms of AS/VS

- Coma vigile
- No recognition of the surrounding
- No reaction to external stimuli
- Reaction on internal stimuli
- Optomotor disturbances (divergent position of the eyes, gaze disturbances)

Symptoms of AS/VS

continued

- Motor disturbances of extremities and trunk (flexed-stretched position of the extremities with fist, rigido-spasticity, hyperreflexia, pyramidal signs)
- Primitive motor patterns (oral, grasping etc.)
- Dysregulation of the vegetative system

Prognosis in traumatic AS/VS

- Can't be made in the first 6 weeks after an acute traumatic brain damage
- Within the first 6 months no decisions can made any about ongoing of active treatment program
- 80% of the patients with an traumatic apallic syndrome develop remission
- Resocialization 25%-30%

Treatment possibilities and outcome

- Modern rehabilitation allows 80% of apallic patients to reach a remission
- Special rehabilitation centers for AS (in Austria: 7 - 44 beds)
- Activating long-term nursing (in Austria: 2 - 28 beds)

  Nursing home (in Austria: 38 - 200 beds)

Similiar situation in Italy, Germany

No treatment wishes for professionals themselves (%)

	PVS/AS	Coma
• Physicians	90%	51%
• Nurses	89%	69%

From Gillick et al. 1993

Ethical principles in decision making in medicine

- Autonomy
- Beneficence
- Maleficence
- Justice

Beauchamp et al, 1979

Withdrawal - Withholding of treatment

It is widely held by philosophers and lawyers, that there is no moral or legal distinction between these two.

Only 20% of UK and 34% of US health professionals agreed that withholding and withdrawing were equivalent, based on the feeling, that when death follows an action, it may seem more culpable.....

Legal basis in the treatment of apallic patients unable to give consent

- Full stage
- Remission stages
- Certain defect stages (severe dementia, frontal lobe syndrome, Klüver-Bucy syndrome, Wernicke aphasia, etc.)

Indipensable need for a solicitor.

Solicitor

"Sachwalter"

- Responsible for general decisions
- Responsible only for financial decisions
- Proposal to the civil court
- Solicitor may be a relative or a lawyer

Decisions to make during the treatment of apallic patients

- Decision for continuation of active rehabilitation
- Decision to interrupt active rehabilitation, transfer to an activating long-term nursing unit
- „Decision" on stopping medical treatment – withdrawal special treatment measurements, strictly regulated in most European countries
- Decision of „end of life" - **active Euthanasia** (no discussion in most European countries)
- Decision on withholding „maximal therapy"– **passive Euthanasia** – physician's responsibility

Euthanasia

Eu-Thanatos – good death

Active Euthanasia

Euthanasia without expression of free will fromy patient, without the knowledge of the patient (Austria: assassination §75 StGb).

Free willing Euthanasia performed by a physician or medical personal (Austria: criminal act §77 StGb).

Assisted suicide (Austria: criminal act §78 StGb).

Passive Euthanasia - included in Hippocratic Oath.

Decision to interrupt the running treatment in almost dying and dying patients, by continuation of all care (nutrition, liquid support, basic medical support, etc.)

Withholding of "maximal therapy" in case of complications in severe and untreatable conditions.

Forced Euthanasia

"Drittes Reich"

Ethnical based (Massai)

Consequence of economical measurements in medicine (refuse to treat old aged people, untreatable cancer patients, etc.)

Decision for withholding „maximal therapy"

Decision made by treating physician considering certain facts:

- Objective criterias (diagnosis, prognosis, patients disposal)
- How the patient himself would decide in this situation
- Solicitor and family

Summary

- Every patient with traumatic brain injury needs an acute treatment and in most cases (moderate, severe, to severest) a neurorehabilitation
- Patients in severest traumatic brain injury have to be treated as if fully recovery is possible
- Apallic patients need a special treatment in acute phase (ICU) and a special neurorehabilitation program in specialized center for apallic patients
- Final prognostic statement not possible before 6 months past the acute accident
- End of life decision in most of European countries out of discussion – **active Euthanasia**
- **Passive Euthanasia** due to withholding of "maximal therapy" is acceptable