2.4
Brain Computer Interfaces (BCI) in Neurological Rehabilitation

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An overview of experimental clinical studies in BCI shows that non-invasive BCIs using various EEG measures is extremly useful and efficient for brain communication in locked-in patients, particularly with amyotrophic lateral sclerosis. First attems to apply BCI for communicating with the completely locked in and vegetative state are promising but inconclusive. A new approach using classical conditioning of brain responses for the completely locked in is demonstrated.
Considerable progress was made in movement reconstruction with BCI in chronic stroke without redidual movement. An EEG-MEG based BCI system using several robotic devices for hand and arm movement and sensorimotor rhythm (SMR) as the guiding signal for the neuroprosthetic device showed significant recovery of arm and hand movement in one third of the patients but difficulties of generalisation to real life. New approaches adding functional electrical stimulation of hand muscles and tDCS for learning are introduced.
Finally metabolic BCI with real time functional magnetic resonance imaging (rt-fMRI) and Near Infrared Spectroscopy (NIRS)BCI has shown considerable success for the control of schizophrenic symptom sand
criminal psychopathy. However long-term improvement was demonstrated only in Attention deficit Disorder and itractable focal epilepsy using neurofeedback of slow cortical brain potential as BCI.
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Panel Session 3 - Neurological Long Term Rehabilitation in TBI

3.1
Introductory comments to Neurorehabilitation after TBI: Biomechanics on TBI

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Understanding the biomechanic aspects in relation to traumatic brain injury simplifies the comprehension of lesion development. Two basic physical factors play an important biomechanic role, the speed v and the acceleration b (b = v2/2s), where s is the deceleration distance. At constant speed of v, b can change to a great extent, if the deceleration distance becomes very small. In the impact moment, the head bone undergoes an elastic deformation. Only if this force in combination with the form of object exceeds the rather individual breaking strength of the skull bone fracture occurs. Location of structure is dependent mostly on the form of impact. At the impact pole and at the counter pole damages occur on the brain surface. Typical pattern of lesions might be identified dependent on the direction of the impact on the head. A common concept to understand occurring patterns is to distinguish between linear brain injury (Grcevic) and/or rotational trauma (Pudenz - Shelden). Usually a combination of both exists. Principally damages of the brain, the brain vessels and the meninges also result from the direction of the impact on the head. We found an adopted Spatz Scheme (Innsbruck modification) valuable to document the direction of impact. This scheme is simple to use and might be applied already at the accident site.
In this lecture biomechanical aspects of typical impact patterns will be presented. Concise evaluation of this data allows the development of an individual neurorehabilitation treatment plan.

3.2
Neurorehabilitation after TBI

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Different issues have to be classified in neuroscience, the acute neurology with diagnosis and treatment, the neurorehabilitation with the principle aim for re-socialisation and the neurological care after the end of treatment state with the aim of amelioration of quality of life. Generally Neurorehabilitation has do be differentiated in actual neurorehabilitation (TBI, stroke etc.), temporal neurorehabilitation (Parkinson's Disease, MS etc.) and palliative neurorehabilitation (malign brain tumor, ALS etc.).The demands of modern treatment in TBI are the exact neurological diagnosis in the initial phase (mild to severest forms), documentation of the impact to the skull (Spatz- Innsbruck Scale). The acute treatment of severe and severest forms of TBI should be organized in a neurological ICU. Early rehabilitation has to be started immediately in all forms of TBI followed if necessary by a consecutive phase.
Every patient needs an individual neurorehabilitation program, which has to be executed in a special neurorehabilitation center, organized as a therapeutic community.
In the neurorehabilitation the task-specific repetitive concept is the basis as the neurophysiological treatment. For the restoration in motoric deficits the different methods can be performed (Bobath, Janda, Vojta), a combination with special medicaments is obligate. Deficits of the higher brain functions (aphasia, alexia etc.) and of the highest brain functions (frontal lobe symptoms, temporo basal syndrome) needs a special programme. A specific neurorehabilitation has to be established in patients with a traumatic apallic syndrome, if the rehabilitation is organized in a special center 25 % of them can be socially reintegrated. In apallic patients no prognosis is possible within the first 6 weeks, no decision about ongoing of active treatment programme in the first 6 months can be accepted.
Any discussion about a preterm ending of neurorehabilitation is not acceptable, from a neurological point of view and the ethical demands.

SELBST HILFE GRUPPE SCHÄDEL HIRN TRAUMA

BIF European Confederation

Trauma Österreichische Gesellschaft für Schädel-Hirn-Trauma

1[st] TBI-Challenge.eu 2011

February 23rd to 26th, 2011 in Vienna

BIENNIAL INTERDISCIPLINARY CONFERENCE
of the
BRAIN INJURY AND FAMILIES / EUROPEAN FEDERATION (BIF)
In Cooperation with local and European TBI Associations

ABSTRACT BOOK

www.tbi-challenge.eu

Introductory comments to Neurorehabilitation after TBI: Biomechanics on TBI

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1st TBI-Challenge.eu 2011
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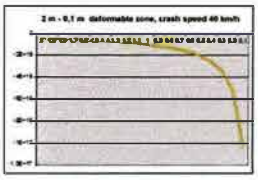
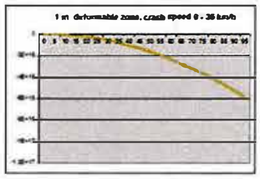
Physics of TBI

- Two physical factors are important:
  speed v
  acceleration b

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

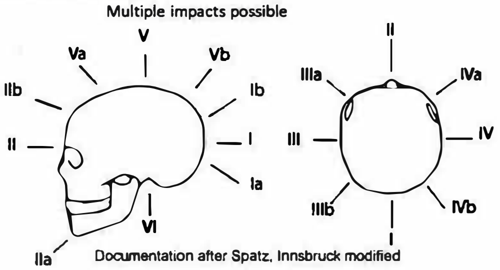
In fact it is $b = \frac{v_a^2 - v_e^2}{2s}$, but v_e^2 is regarded to be 0 = deceleration until complete stop





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

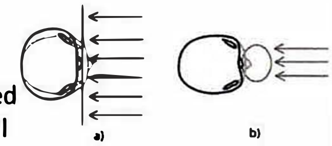
Brain tissue damage depends on
- Direction, form of impact
- Location of impact
- Intensity of the force
- Multiple impacts possible



Documentation after Spatz, Innsbruck modified

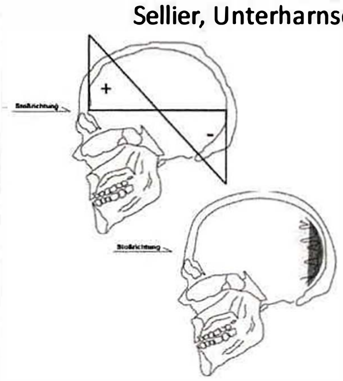
Different types of TBI

- Closed cerebral trauma, sometimes combined with fracture of skull



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

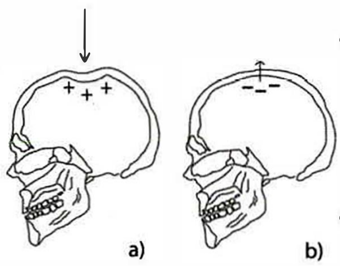
Biomechanics, physical analysis
Sellier, Unterharnscheidt, 1963



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

with permission of K. Jellinger, Vienna

Biomechanics, cavitation trauma



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

Different Types of Brain Trauma
Classification by biomechanical analysis

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

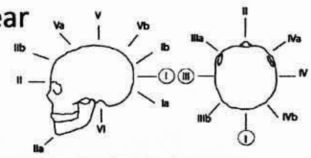
Patterns of cerebral trauma
Acceleration – Deceleration trauma

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

Different forms of traumatic lesions

- Primary lesions (irreversible)
- Secondary lesions (therapeutic battle field)
  - Penumbra (local),
  - Non cerebral functional disturbances: posthypoxic, posthypoxemic, postedemic, circulatroy (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

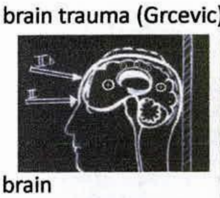
Different Types of Linear Outer Brain Trauma



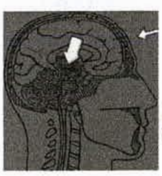
Impact type I   Impact type III

Linear Inner Brain Trauma

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



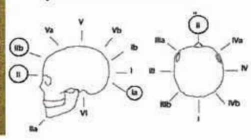
b) Linear inner lower brain trauma (Lindenberg)
lesions brain stem, surrounding brain region
Type V, Va



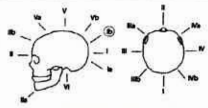
Linear Inner Upper Brain Trauma
Schematic drawing (N. Grcevic)

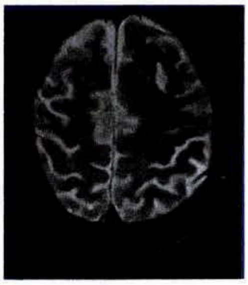
Impact type IIb, Ia, (II)

Main lesions, periventricular



Linear Inner Upper Brain Trauma Type Ib

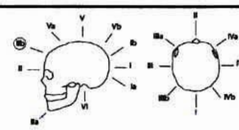


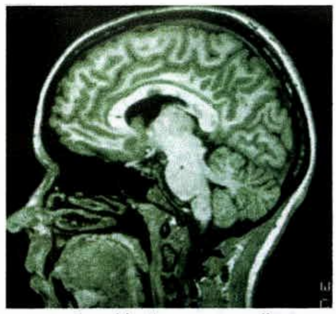


Parasagittal lesion, butterfly type

Lesion corpus callosum, butterfly type

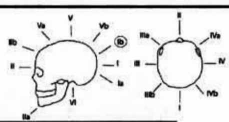
Linear Inner Upper Brain Trauma, Impact Type IIb

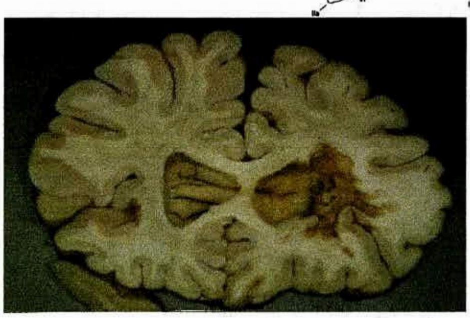




Local lesion corpus callosum

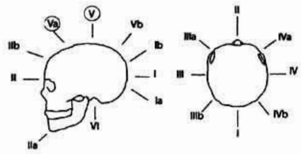
Linear Inner Upper Brain Trauma Type Ib





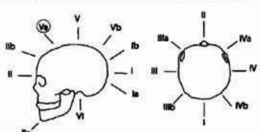
Frontal white matter, periventricular damage

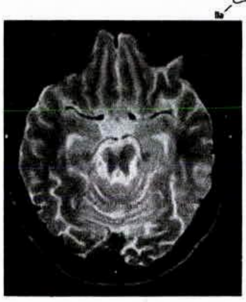
Linear Inner Lower Brain Trauma (Lindenberg) Type V, Va



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

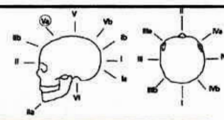
Linear Inner Lower Brain Trauma Type Va, Primary lesions

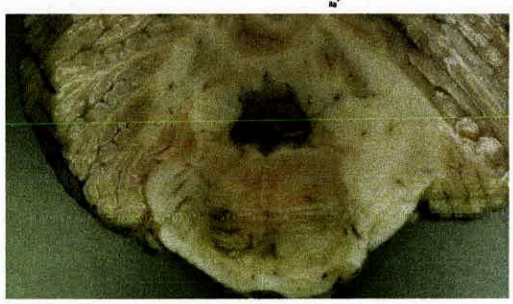




Mesencephalon

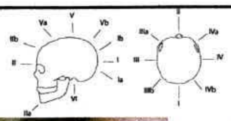
Linear Inner Lower Brain Trauma, Type Va, Primary lesions

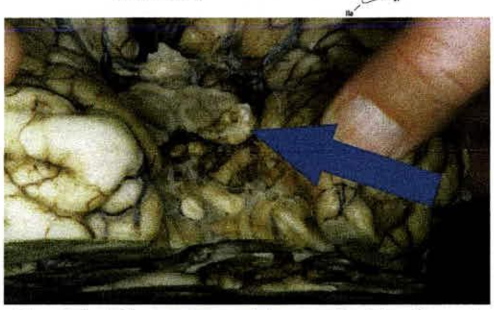




Gliotic lesions with haemosiderin deposition, lower midbrain, pons

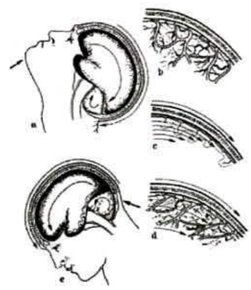
Linear Inner Lower Brain Trauma
Combination with uncal tentorial herniation

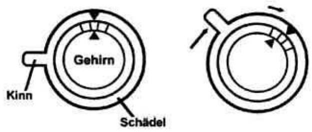




Primary lesion in the upper mesencephalon, secondary lesion after uncal herniation (arrow)

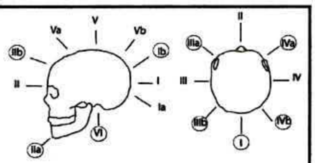
Rotational trauma – Scheme
Pudenz-Shelden





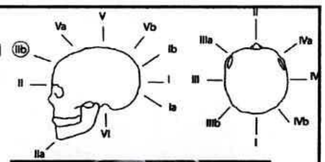
- Laceration (capsula int., basal ganglia)
- Intracerebral haemorrhage (thalamus, hypothalamus)
- Extracerebral haematoma (subdural, epidural)

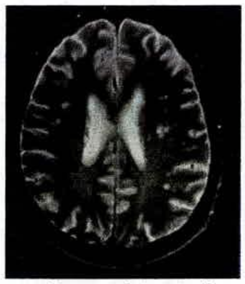
Rotational Trauma
(Pudenz-Shelden)
Type Ia, Ib, IIa, IIb, IIIa, IIIb, IVa, IVb, VI



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

Rotational Brain Trauma Type IIb





White matter lesions, small hematoma

Lesions: basalganglia, capsula interna

Take home messages

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