normal cyclic wake-sleep pattern in 12 subjects. Clinical Outcomes were: 3 deaths; 5 VS patients; 7 minimally conscious state patients (MCS).

Conclusion. Our study describes the polysomnographic EEG patterns in an heterogeneous group of VS patients. We found how it is difficult to consider common scoring criteria both due to clinical situation and environmental conditions. Despite this we could perform conventional scoring in 40% of the patients. Remaining observations revealed different patterns as "dissociated patterns" with the presence of phasic rhythms. We observed that 5 Patients with REMs evolved to a MCS.

P056

Functional Involvement of cerebral cortex in patients with sleep-wake disturbances after traumatic brain injury: a TMS study

S.M. Golaszewski[1,2,5], M. Seidl[1,2,5], A.B. Kunz[1,2,5], F. Caleri[3], P. Lochner[3], S. Cataldo[3], F. Gerstenbrand[4,5], E. Trinka[1], R. Nardone[1,3]

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[3] *Franz Tappeiner Hospital, Meran, Italy*
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[5] *Karl Landsteiner Institute of Neurorehabilitation and Space Neurology, Vienna, Austria Karl Landsteiner Inst. of Neurorehabilitation a. Space Neurol., Vienna, Austria*

Objective: Sleep-wake disturbances (SWD) are common after traumatic brain injury (TBI); in particular, chronic excessive daytime sleepiness (EDS) is a major, disabling symptom for many patients with TBI. The pathophysiological mechanisms remain unclear. Transcranial magnetic stimulation (TMS) represents a useful complementary approach in the study of sleep pathophysiology. We aimed to determine in this study whether post-traumatic SWD are associated with changes in excitability of the cerebral cortex.

Participants, Materials/Methods: TMS was performed 3 months after mild to moderate TBI, in 11 patients with subjective excessive daytime sleepiness (defined by the Epworth Sleepiness Scale ≥ 10), 12 patients with objective EDS (as defined by mean sleep latency < 5 on multiple sleep latency test), 11 patients with fatigue (defined by daytime tiredness without signs of subjective or objective EDS), 10 patients with post-traumatic hypersomnia "sensu strictu" (increased sleep need of >2 h per 24 h compared to pre-TBI), and 14 control subjects. Measures of cortical excitability included central motor conduction time, resting motor threshold (RMT), short latency intracortical inhibition (SICI) and intracortical facilitation to paired-TMS.

Results: In the patients with objective EDS and hypersomnia, RMT was higher and SICI was more pronounced than in control subjects. In the other patients all TMS parameters did not differ significantly from the controls.

Conclusions: Similar to that reported in patients with narcolepsy, the cortical hypoexcitability may reflect the deficiency of the excitatory hypocretin/orexin-neurotransmitter system.
A better understanding of the pathophysiology of post-traumatic SWD may also lead to better therapeutic strategies in these patients.

P057

The role of functional MRI in diagnosing severe chronic disorders of consciousness

S. Golaszewski[1,2,5], M. Seidl[1,2,5], A.B. Kunz[1,2,5], M. Kronbichler[3], J. Crone[3], R. Nardone[1,4], E. Trinka[1], F. Gerstenbrand[5,6]

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Objective: Accurate diagnosis of severe chronic disorders of consciousness (DOC) after TBI is essential for clinical and rehabilitative care and decision-making. Neurobehavioral tests, which rely on the patients' intellectual and motor ability to communicate, are the most widely used diagnostic tools, since their advantage over clinical assessment has been validated. However, with the emergence of modern neuroimaging methods, especially functional MRI, objective physiological markers for assessing the state of consciousness are available in specialized clinics. They are, however not fully integrated in clinical routine, because their benefit has yet to be determined.

Participants, Materials/Methods: 15 patients in apallic syndrome (AS) and 5 patients in minimally conscious state (MCS) after TBI and other etiologies were examined with somatosensory, auditory and event related paradigms in fMRI and evoked potentials (EP). The findings were compared to the neurobehavioral diagnosis and it was analyzed, if the additional information from fMRI and EP confirmed or questioned the diagnosis.

Results: 3 out of 15 patients in AS showed fMRI activation in event related paradigms, suggesting that patients are in MCS or even better.

Conclusion: Uncertainty in diagnosis still exists even with well-established diagnostic assessment scales. As long as internationally accepted guidelines for assessing patients with chronic DOC do not exist, every single diagnostic modality available in each clinical setting should be performed, to minimize diagnostic error and to find ways, in terms of perceptive channels, to approach the patients. FMRI has the potential to bring diagnostics in chronic DOC forward to the next level.

P058

The "Extended Locked-in syndrome"

Gerstenbrand[1,2], S.M. Golaszewski[2,3,4], M. Seidl[2,3,4], A. Kunz[2,3,4], R. Nardone[3,5], G. Bauer[1], E. Trinka[3]

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[2] *Karl Landsteiner Inst. of Neurorehabilitation a. Space Neurol., Vienna, Austria*
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[5] *Franz Tappeiner Hospital, Meran, Italy*

Objectives: Locked-in syndrome is one of the most devastating neurological conditions. However, despite thorough description of the condition and its clinical appearance, the classic Locked-in syndrome, which is defined as quadriplegia, only vertical eye movement and blinking possible with preserved cognitive abilities, seems to be infrequently present. This syndrome is also referred to as bilateral ventral pontine syndrome, which in respect neuroanatomically explains the symptomatology. Since MRI verified isolated damage to the pons poses the finding in this certain case, the question arises, how the symptomatology increases, if additional lesions are found in cranial brain areas. The aim of the study is to describe in detail different clinical syndromes and to relate them to different patterns of structural damage in 3T MRI.

Participants, Materials/Methods: Five patients with brainstem infarction and different patterns of structural injury and clinically in a state of unresponsive wakefulness are investigated with structural 3T MRI.
Results: Clinical and MRI results are presented in great detail and it is discussed how clinical appearance and imaging results relate to each other. The question will be approached if it is useful to differentiate several types of Locked-in syndrome and how akinetic mutism and parasomnial syndromes connect in addition.

Conclusion: Especially since special academic emphasis is placed

on research of coma and chronic disorders of consciousness, which typically centres on neocortical structures, we take a look upside down from the brainstem to higher order brain areas and propose a hierarchical scheme of consciousness beginning from Locked-in syndrome to vegetative state.

P059

The functional anatomy of motor imagery in stroke

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Motor imagery (MI) refers to the ability to imagine the performance of a movement without actually executing it. The neural correlates of this cognitive process are tightly coupled with the cortical motor control network. Therefore MI has a therapeutic potential for patients with motor deficits after ischemic stroke. We studied 17 patients after cerebral ischemic stroke (subacute stage) and 12 healthy subjects using functional Magnetic Resonance Imaging during motor imagery and actual performance of isometric grip force movements (5N). All patients had a paretic hand, but were able to perform the grip force movement. We were particularly interested in alterations of interhemispheric balance of activity in stroke patients. For this purpose analysis of regional activations was performed by calculating laterality indices (LI) to assess hemispheric distribution of pre-specified motor areas.
Laterality index (LI) revealed a more balanced cortical activity in MI for both controls and patients in premotor cortex compared to movement execution and a trend towards a shift in contralesional activity in stroke patients. Our results indicate a preserved ability of patients to activate the preparatory motor areas associated with MI. This lends additional support to the use of MI as an adjunct rehabilitative approach for improving functional recovery in stroke.

P060

Two years outcome and the level of professional reintegration after severe traumatic brain injury

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Introduction: The interest concerning the level of professional reintegration and functional outcome of patients with traumatic brain injury (TBI) has increased during the last years.
The aim of the study was to evaluate the level of professional reintegration of patients with severe traumatic brain injury (STBI) after 12 and 24 months.

Methods and subjects: A total of 51 STBI patients (Glasgow Coma Scale GCS </= 8 points for at least 24 hours) underwent a multidisciplinary early rehabilitation treatment until they were discharged from hospital and local ambulatory care was deemed sufficient. The follow-up examination took place 12 and 24 months after the STBI.

Results: Data revealed a high level of independence in activities of daily living (mean Barthel Index after one year 92.7 points, after two years 93.7 points). After one and two years, 74.5% and 80.4% of the patients, respectively, were completely independent of need for care. Nevertheless, more than half of the patients had sensorimotor, behavioural, speech, visual and/or auditory disturbances. Return to work rates improved between one and two years after trauma, as evidenced by the rate of patients being back to full time work at one year (n= 14, 28%) and two years (n=20, 40%) post-STBI. Return to work rates improved between one and two years after trauma, as evidenced by the rate of patients being back to full time work at one year (n= 14, 28%) and two years (n=20, 40%) post-STBI; although, none of these changes reached statistical significance.In those subjects who were not or only very restrictedly able to work, behavioural and speech deficits were significantly more frequent.

Discussion: In summary, there are still changes in both impairment and disability related areas between one and two years post-STBI, but the degree of improvement is variable depending on the area being considered. Clinicians should remain aware of the fact that modulation of impairment and disability appear to continue well beyond one year post-STBI which may impact on decisions regarding the provision and intensity of further rehabilitation efforts.

Conclusion: Behavioural and speech deficits seem to represent the major cause that hinders professional reintegration. Rehabilitation therapy therefore should be specifically directed to improve these deficits.

P061

Nonverbal therapeutic techniques in rehabilitation of individuals surviving traumatic and non-traumatic brain injuries

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Nonverbal therapeutic techniques (art-therapy, dance and movement therapy- DMT, music therapy) serve as a psychotherapeutic use of a movement, art gesture or sound to enable process for improving the emotional, cognitive, social and physical integration of the organism. Patients after brain injury (BI) suffer from problems in all those areas. Presentation focuses on a specific movement and art features of BI patients as a consequences of an organic brain trauma, as well as resulting psychological and social problems. Careful diagnostic process guides us to targeted use of a movement or art techniques to influence emotions, cognition or motoric area. The therapeutic aim in DMT is to explore, sense and recognize owns body, body image, body borders and movement repertoire in connection to emotions. Through artistic creation we explore conscious and unconscious processes and work with them verbally or nonverbally. DMT and art-therapy works with regressions to a lower movement and drawing patterns. Group therapy enables communication and socialization in the group, work on self-esteem, self-trust and self-admitting.

P062

Communicative deficit in traumatic brain injured patients: Analysis of errors

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Introduction/Objectives: The aim of the present study is to provide an in-depth description of the communicative errors made by TBI patients, a topic often overlooked in the literature.

Participants, Materials/Methods: 30 TBI patients plus an equal number of normal matched controls took part in the experiment. They were tested on the Linguistic and Extralinguistic scales of the Assessment Battery for Communication (Sacco et al. 2008, *Journal of Cognitive Science*, 2, 111-157), which consists of a series of pragmatic tasks involving the comprehension or production of different kinds of communicative acts (i.e. direct and indirect communicative acts, irony and deceit). The participants' answers were evaluated as correct or incorrect. Incorrect answers were then further rated as totally or partially correct corresponding to intermediate levels of comprehension. This was the case when, for instance, the patient correctly understood the literal meaning of the utterance but not the speaker's meaning,

S-13-004
Contribution of abdominal obesity and genetic markers of obesity to risk of ischemic stroke, intracerebral hemorrhage and transient ischemic attack
Y. Winter, Marburg, Germany
A. Scherag, J. Linseisen, S. Rohrmann, A. Hinney, M. Neumaier, P. Ringleb, R. Dodel, J. Hebebrand, T. Back

S-13-005
Nuclear gene mutations in chronic progressive external ophthalmoplegia with multiple deletions of mitochondrial DNA
S. Jackson, Dresden, Germany
Schaefer, D. Leupold, S. Clodius, K. Witte, M. Weinhold, B. Cruno, H. Reichmann

S-14 SYMPOSIA

13.00–14.35 h **Auditorium 2**

Sleep Symposium

Chairs D. Hermann, Essen, Germany
R. K. Chaudhuri, London, United Kingdom

S-14-001
Disordered sleep in parkinsons's disease
R. K. Chaudhuri, London, United Kingdom

S-14-002
Disordered sleep in stroke
D. Hermann, Essen, Germany

S-14-003
Disordered sleep in infection and inflammation
A. Schuld, Ingolstadt, Germany

S-14-004
Functional involvement of cerebral cortex in patients with sleep-wake disturbances after traumatic brain injury: a TMS study
S. Golaszewski, Salzburg, Austria
M. Seidl, A. Kunz, F. Gerstenbrand, E. Trinka, R. Nardone

S-14-005
Cognitive disorders in patients with chronic obstructive pulmonary disease
U. Kolcheva, St.Petersburg, Russia

O-02 ORAL PRESENTATIONS

15.00–16.30 h **Auditorium 2**

Oral Presentations 2

Chairs O. Bajenaru, Bucharest, Romania
D. Bereczki, Budapest, Hungary

O-02-001
Lyme borreliosis-associated cerebral vasculitis with cerebral ischemia: 11 cases from an East German region
T. Back, Arnsdorf, Germany
S. Grünig, U. Bodechtel, K. Guthke, D. Khati, R. von Kummer

O-02-002
A symtomatic carotid artery stenosis. Should we stop this procedure and/or should we continue?
D. Bartko, Ruzomberok, Slovakia
Z. Gpmbosova, I. Combor, F. Rusnak, L. Danihel, J. Kodaj, V. Sefranek, K. Zelenak

O-02-003
The extended locked-in syndrome
S. Golaszewski, Salzburg, Austria
M. Seidl, A. Kunz, R. Nardone, E. Trinka, G. Bauer, F. Gerstenbrand

O-02-004
Self management of headache: a cross-sectional survey in the general public of islamabad
S. Ghumman, Islamabad, Pakistan
M. Nadeem, M. Umer Azeem, A. Javed Nawaz, Z. Hassan Khan, R. Meena Kakar, A. Ali Khan

O-02-005
The role of functional MRI in diagnosing severe chronic disorders of consciousness
S. Golaszewski, Salzburg, Austria
F. Gerstenbrand, M. Seidl, A. Kunz, R. Nardone, E. Trinka

O-02-006
Neurological capacities, their utilization and workload on neurologists in Hungary
D. Bereczki, Budapest, Hungary
A. Ajtay

43rd International Danube Neurology Symposium 2011

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6 – 8 October 2011
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www.danube2011.org

The extended Locked-in Syndrome

S. Golaszewski [1], M. Seidl [1], A. Kunz [1], R. Nardone [2], E. Trinka[1], G. Bauer[3], F. Gerstenbrand[4]

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The term „Locked in Syndrome (LIS)" was coined 1966 by Plum and Posner to describe a state of quadriplegia and anarthria with preserved consciousness [1]. It is most often caused by ischemic stroke or hemorrhage, affecting the corticospinal, corticopontine and corticobulbar tracts in the brainstem [2], however, also midbrain infarctions of the bilateral cerebral peduncles causing LIS has been reported [3,4]. Other causes of LIS include trauma [2], pontine abscess [5], brainstem tumors[6], central pontine myelinolysis, toxins and heroin abuse [7]. 1979 Bauer et al. classified three varieties of LIS [8]: First, classical LIS presenting with total immobility except for vertical eye movements and blinking, second, incomplete LIS, if any other movements are present and third, total LIS with total immobility, including all eye movements, combined with signs of undisturbed cortical function in the EEG.

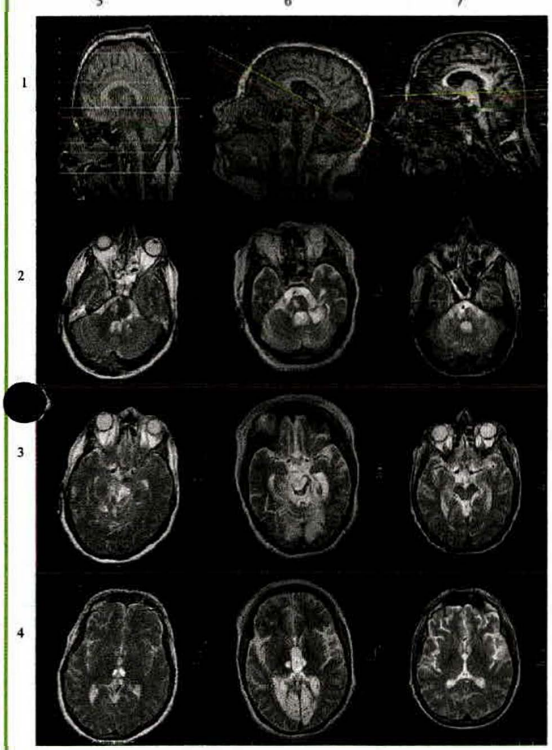
In the following, we describe a fourth variety, 2 cases of „extended Locked in Syndrome" (patients W.B. and S.M.), where patients additionally to the classical or total LIS also suffer from other symptoms. To show anatomical and clinical differences we also present one case of classic LiS (patient H.D.). All patients suffer from brain damage following a basilar artery thrombosis.

The extent of brain tissue damage, in order to meet the diagnostic criteria for classic LiS has to be isolated to the ventral part of the pons, comprising the corticospinal and corticobulbar tracts, as well as the paramedian pontine reticular formation (PPRF), which is in particular responsible for horizontal eye movements and saccades. Vertical eye movements and blinking are controlled by the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF), which is located cranial in the pontomesencephalic junction. Therefore involvement of paramedian mesencephalic structures can cause vertical gaze paralysis. The additional involvement of riMLF in brainstem damage leads to the clinical presentation of total LiS. Otherwise, the sparing of PPRF and riMLF results in incomplete LiS presenting without oculomotor disturbances. Patient H.D. suffers from a classic LiS.

Discussion:

The main clinical relevant differences between a classic LiS and an extended LiS lies on the one hand in a variety of additional extra pontine brain lesions, on the other hand in presented symptoms following these lesions: Frequenty occuring symptoms in extended LiS are hypersomnia syndroms, frontal release signs, thalamic posturing of hand and/or feet. Rarely an akinetic mutism may be present.

Extra pontine brain lesions may frequently occur in mesencephalic, thalamic and cerebellar brain structures. Also involvement of occipital, temporal brain regions is possible – depending on varieties of the vertebro-basilar artery blood supplying system.



MRI images (Sagittal T1w, axial T2w):
In the vertical column, there are MRI images of each patient at different levels:
1: Sagittal plane
2: Pons level – middle cerebellar peduncles
3: Midbrain level
4: Thalamus
5: Patient W.B.
6: Patient S.M.
7: Patient H.D.

Patient W.B.: extended LiS

Clinical characteristics: Divergent bulbi, arreagible pupils, anisocoria r>l, no visual pursuit, OCR and corneal reflex missing. Flaccid quadriparesis. Distal flexor contractures, no reaction to noxious stimuli. Babinski's sign present.
No communication with patient possible – additionally the patient presented with a hypersomnia syndrom
Imaging findings: Gliotic degeneration of the upper 2/3 of the brainstem paramedian bilaterally. Involvement of midbrain structures and dorsomedian thalamic nuclei bilaterally Over time atrophy of cerebellum, brainstem and corpus callosum.

Patient S.M. extended LiS

Clinical characteristics: Divergent bulbi, arreagible pupils, anisocoria r>l. Missing OCR, corneal reflex and gag reflex. Flaccid quadriplegia, Babinskis's sign negative.
Distinct frontal release signs: orbicularis oris reflex, sucking reflex present.
Additionally the patient shows a hypersomnia syndrome and a bilateral thalamic hand: flexion in the MCP joint, extension in the distal joints.
Imaging findings:
Gliotic transformation on the right side of the pons (image 2/6) and the left middle cerebellar peduncle. Nearly the whole cross section of the midbrain affected (image 3/6) Bilateral dorsomedian thalamic nuclei involved, as are bilateral occipital, mesiotemporal and cerebellar brain regions.

Patient H.D.: classic LiS

Clinical characteristics: Round , isocor pupils, reactive to light . OCR and corneal reflex present. No paralysis of eye muscles.
Spontaneous movement of the right upper and lower extremity. Retraction after noxious stimuli. Plegia of the left upper and lower extremity.
Communication possible with the use of eye movements and movement of the right side extremities.
Imaging findings: Isolated damage to the ventral pons (image 2/7). Sparing of midbrain and thalamic structures. However, gliotic degeneration in the right cerebellar hemisphere and in the right upper and middle cerebellar peduncle.

References:
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Karl Landsteiner Institute of Neurorehabilitation and Space Neurology

The Extended Locked-In Syndrome

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St. Golaszewski[3)], M. Seidl[3)], A. Kunz[3)], E. Trinka[3)], Salzburg
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43rd International Danube Neurology Symposium
6 – 8. Oktober 2011
Dresden, Germany

Symptoms of Locked-In Syndrome

- No possibility to communicate with surrounding except for blinking morse code
- Consciousness and perception fully maintained
- Total paralysis of all extremities, trunk, neck and motor brain nerves
- Eye opening and vertical eye movements possible
- Impairment of swallowing
- Spontaneous respiration possible
- Alpha-EEG

Etiology of Locked-In Syndrome Lesion in Pons

- Infarction caused by basilar thrombosis
- Hemorrhage
- Encephalitis
- Tumor
- Traumatic lesion

Appearence of LiS

- **Synonym:** Ventral pontine syndrome
- **Clinical signs:** Anarthria, Quadriplegia, intact cognitive abilities. Blinking and vertical eye movement possible.
- **Etiology:** typically: basilar artery thrombosis, brainstem hemorrhage, rare: brainstem neoplasm or metastasis.

Graduating LiS (Bauer et. al 1979)

- Classic LiS
  - » *brain lesions*: ventral pons
  - » symptoms: quadriplegia, anarthria, unimpaired consciousness, vertical eye movement, blinking possible
- Complete LiS
  - » *brain lesions*: ventral pons plus rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF), located cranial in the pontomesencephalic junction
  - » symptoms: classic Locked-In Syndrome plus no possible movement of eyes or eyelid
- Incomplete LiS
  - » *brain lesions*: variable brainstem lesions
  - » symptoms: additional voluntary muscular movement

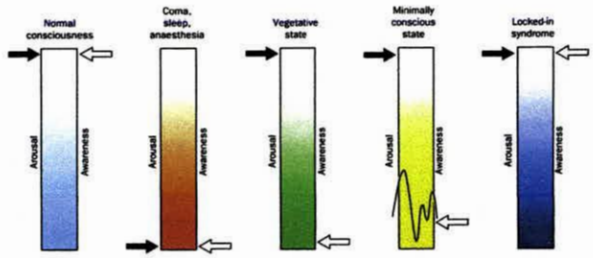
Different Types of LIS (after Bauer et al, 1979)

- According to neurological symptoms
  - – Classical Locked-in syndrome
  - – Incomplete Locked-in syndrome
  - – Total Locked-in syndrome
- According to time course
  - – Chronic Locked-in syndrome
  - – Transient Locked-in syndrome

Differential Diagnoses

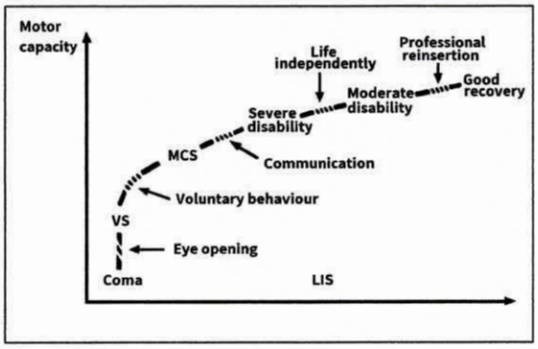
- Akinetic mutism
- Hypersomnia
- Parasomnia
- Apallic syndrome, Remission states

Remission of a Coma State after Laureys et al



Arousal and awareness, the 2 components of consciousness in coma, vegetative state, minimally consciousness state and Locked-In syndrome

Restauration of a Coma State after Laureys et al



Acute coma, passing, vegetative state, minimally consciousness state, good recovery

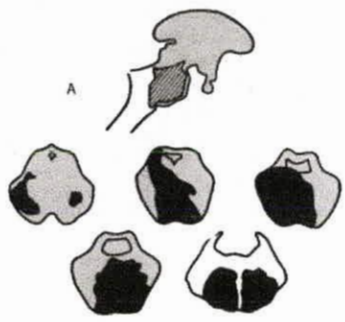
Different Forms of Disturbancies in Consciousness

- Acinetic mutism (Cairns et al., Skultety)
  - Lesion region 3rd ventricle, periaqueductal
- Sopor
- Stupor (Plum, Posner)
  - Lesion intralaminar nucleus thalami
- Hypersomnia (Jefferson)
  - Lesions mesodiencephal
- Parasomnia (Facon et al.)
  - Lesion periaqueductal

Changement in Consciousness

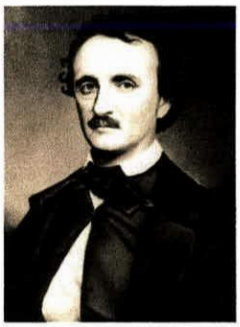
- Acinetic mutism (Cairns et al, Skultety)
  - Disturbance in the initiation of spontaneous and intentional movement
  - Awareness undisturbed
- Sopor
  - Abnormal deep sleep not to get rid
- Stupor (Plum, Posner)
  - Deep sleep, unresponsiveness, temporarily arousable
- Hypersomnia (Jefferson)
  - Dormancy, continuously, not arousable
- Parasomnia (Facon et al)
  - Permanent dormancy, awakes by himself after months
  - Not identical with description/classification of sleep disorders

Differnt Pons Lesions causing LIS



Quelle: F. Plum, J.P. Posner: Diagnosis of Stupor and Coma

Edgar Allen Poe



*January 19th 1809 in Boston, Massachusetts, USA

† October 7th 1849 in Baltimore, Maryland

Was liberated from a coffin already prepared for his funeral after his loud screaming was heared.

Possibility of extended Locked-in syndrome

Nikolai Wassiljewitsch Gogol

*March 20th, 1809 in Welyki Sorotschynzi, Oblast Poltawa, Ukraine;

† February 21st, 1852 in Moscow

In the 1840's developed a schizophrenic pschychosis

Has been burried anabioticly
Has been found completely distorted by his exhumination
extended Locked-in syndrome

The extended Locked-in Syndrome

- combines *symptomes of LiS* <u>with</u> *symptoms of:*
  - Akinetic Mutism
  - Hypersomnia
  - Parasomnia
  - Apallic syndrome/ Remission States

Extended LIS

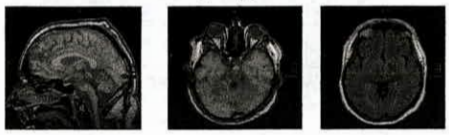
The fate of the Belgian patient Rom Houben caused great commotion, not only in neurological circles but also in the public opinion. The patient, totally paralyzed, was diagnosed as Vegetative State/Apallic Syndrome. Only 23 years later Stephen Laureys discovered that he was not in a coma vigile, but in an alert condition. The diagnosis LIS was clarified.

The consequence of this case and some other similar patients opened the discussion about the classification of LIS and the possibility of a combination with extended symptoms.

Exact clinical analysis and additional examinations are demanded with the aim to search for symptoms caused by lesions in the surrounding region.

Two of five patients of the University Clinic Salzburg with possible Extended LIS are presented.

40 year old male, LIS
Acinetic Mutism / Hypersomnia

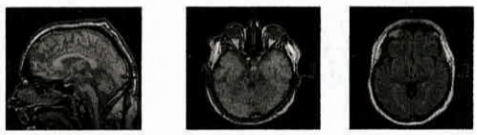


Case 1

Cystic-gliotic degeneration of the bilateral paramedian pons and mesencephalon. Bilateral paramedian thalamus affected as well (dorsomedial nuclei, see third picture). Dorsally, the degeneration of the brainstem reaches the fourth ventricle. Small islands of preserved tissue. High degree of atrophy of the cerebellar peduncles, the cerebellum and the medulla oblongata. Degeneration of bilateral temporomesial structures, especially the parahippocampal gyrus.

Negligible small chronic vascular lesions in supratentorial white matter. The corticospinal tract is affected at a mesencephalic and pontine level.

40 year old male, LIS
Acinetic Mutism / Hypersomnia

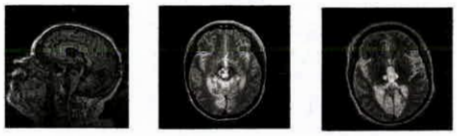


Case 1

After admission to the hospital, a CT scan with i.V. contrast showed occlusion of the distal part of the basilar artery. The posterior cerebral arteries were supplied by the carotid arteries.

A total LIS developed, symptoms of hypersomnia were registered together with signs of an acinetic mutism. The patient showed severe optomotoric disturbances. After six months primitive motor patterns were observed.

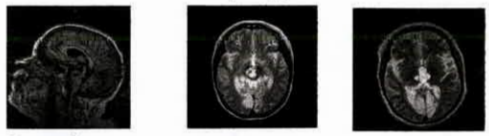
29 year old female, LIS
Acinetic Mutism / Hypersomnia



Case 2

Cystic-gliotic degeneration of the bilateral paramedian pons and mesencephalon. The right side being more affected. Bilateral paramedian thalamus affected as well (dorsomedial nuclei, see third picture). Infarctions in the posterior cerebral artery territory bilaterally, as well as in the left middle cerebellar peduncle and the left cerebellum. Expansion of the third ventricle. The corticospinal tract seems to be completely cut at the level of the pontomesencephalic junction. Slight atrophy of the posterior part of the corpus callosum, the cerebellum and brainstem.

29 year old female, LIS
Acinetic Mutism / Hypersomnia



Case 2

In the initial state a basilar thrombosis was found. Due to wide spread lesions in the initial CT scan and multiple vascular occlusions in DSA, no intra-arterial therapy was performed. A total LIS has developed with symptoms of a lesion accentuated in the right thalamus region. Patient showed hypersomnia and signs of an acinetic mutism, a Bed-Rest Syndrome has developed.

Extended LIS

Intensive examination and continuous observation of patients with Locked-In Syndrome discovered additional symptoms in addition to the basic picture of:

- Acinetic mutism
- Hypersomnia
- Thalamic symptoms

After further detailed examination of LIS-patients other symptoms can be expected as:

- Parasomnia
- Defect state of Vegetative State/Apallic Syndrome

The same examination activities must be carried out in patients with a vegetative state / apallic syndrome for additional symptoms clarifying patients with Vegetative State/Apallic Syndrome and its different remission forms as well as patients with a Minimally Conscious State (MCS).