

**THE OFF-LINE BRAIN – IS THERE SUCH A THING?
APALLIC SYNDROME AND LOCKED-IN SYNDROME**

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Coma Vigile (Wachkoma), a special form of a coma, is the leading symptom of the Apallic Syndrome/Vegetative State. The patient is conscious, but without higher and highest brain functions and shows more or less uniform neurological symptoms.

Pathophysiologically the Apallic Syndrome is comparable to the physiological brain functions of a new born or a young child. An Apallic Syndrome after acute severe brain damage shows a typical course going through from an initial state to a full state. In a great number of patients, a remission is following (Gerstenbrand, 1967). In the remission state the disturbed consciousness in form of a coma vigile shows a reintegration, motor deficits and sensory dysfunctions are restored, higher and highest brain functions, the cognitive abilities, are redeveloping. The Apallic Syndrome after a progressive brain process (Alzheimer Disease etc.) shows a disintegration of all brain functions to the end stage of an Apallic Syndrome (Vegetative State).

The Apallic Syndrome/Vegetative State must not be equated with an Off-line Brain, but can be compared with a partly On-line Brain. The irreversible Off-Line Brain corresponds with the Brain Death Syndrome. The term Coma cannot be equated pathophysiologically with an Off-Line Brain, because of the various accompanying symptoms, which are not recognized.

In contrast to the Apallic Syndrome/Vegetative State the classical Locked-In Syndrome shows only a loss of the motor functions. The patients are conscious with sleep/wake rhythm and full active sensory functions. In the extended Locked-In Syndrome based on enlarged lesions in the mesodiencephalic region, sometimes including parts of the thalamus, symptoms of a stupor, parasomnia, hypersomnia, acinetic mutism and thalamic symptoms can be found.

Only the irreversible Brain Death, the total Brain Break Down, can be called Off-Line Brain.



Karl Landsteiner Institute
of Neurorehabilitation and
Space Neurology

The Offline Brain - is there such a thing? Apallic Syndrome and Locked-In Syndrome

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**42nd Danube Symposium for Neurological Sciences
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„The Online Brain“ Brain functions

- Control center of the body
- Responsible for consciousness
- Processing all incoming data, sensory feelings, etc.
- Acts as an operator by sending messages from all over the body to their proper destination
- Controlling of outgoing messages
- Operating all body movements
- Archive and memory of life experience

Consciousness Medical overview

- Awareness
- Alertness
- Wakefulness
- Attention
- Arousal
- Responsiveness
- Subjectivity

„Offline Brain“ Coma

Definition after Brihaye et al.

- Coma is defined as the pathological status of a patient who cannot be aroused to a wakeful state and whose eyes are continuously closed and do not open on command or on receipt of nociceptive stimuli.

Brihaye J, Frowein RA, Lidgren S, et al. Report of the meeting of the WFNS Neuro-traumatology Committee, 1. Coma-Scaling. Acta Neurochir 1978;40:181.

„Offline Brain“ Coma

Definition after Plum and Posner

- Deep unarousable unconsciousness

Plum, F, Posner J.B., The Diagnosis of Stupor and Coma. F.A. Davis Company, Philadelphia, 3rd Edition, 1980,

„Irreversible Offline Brain“ Brain Death

- The term *brain death* is defined as "irreversible unconsciousness with complete loss of brain function," including the brain stem, although the heartbeat may continue.

Encyclopedia of Death and Dying
<http://www.deathreference.com/BI-Ce/Brain-Death.html>

Brain Death Definition after Shewmon

Brain Death is stated in patients where continuing treatment of this patient does not include any hope of regaining any level of brain function. A continuation of therapeutic measures in brain death is neither in the interest of the patient nor ethically permitted. To treat a living corpse is unethical, it reduces a human being „ to a mere collection of organs“ Shewmon (1998).

„Chronic Brain Death“: Brain dead patients still „alive“: 56 patients for more than 1 month, 7 patients over 6 months, for more than 1 year, 1 patient 14,5 years
Shewmon (1998)

Brain Death

Differential diagnoses

- apallic syndrome / vegetative state
- locked-in Syndrome

Symptoms of Apallic Syndrome

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

Etiology of Apallic Syndrome

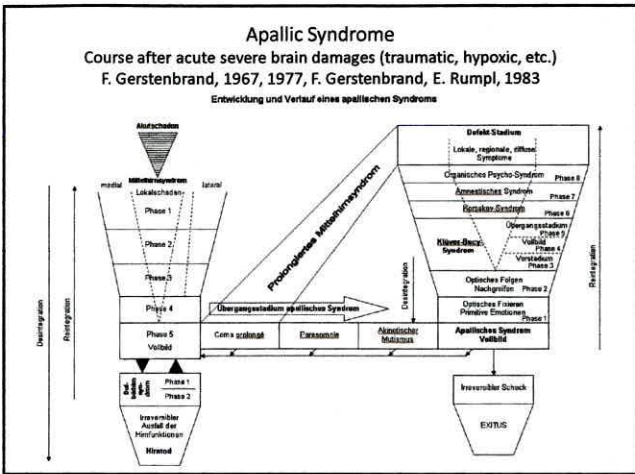
1. After acute, severe brain injuries
TBI, encephalitis, hypoxia, malignant stroke etc.
– Possibility of remission
2. After progredient, diffuse brain processes
CJD, M. Alzheimer, M. Pick, Chorea Huntington, etc.
Final stage
– Remission not possible
3. Intoxication
 - 3a Acute
Exogenous (neuroleptics etc.)
Endogenous (hepatic, uremic etc.)
– Full remission possible
 - 3b Chronic
Exogenous (Minamata disease etc.)
Endogenous (hepatic, thyreotoxic etc.)
– Partial remission possible

Historical background to the diagnosis of Apallic Syndrome

- Coma:
- State of deep unarousable unconsciousness Plum, Posner 1972
 - Prolonged unconsciousness French, 1952
 - Coma prolongé three stages Vigouroux et al. 1964
 - Coma carus
 - Coma avec stabilisation des phénomènes végétatifs
 - Coma, phase sortie de l'état comateux

Coma prolongé, three stages Vigouroux et al. 1964

- Coma carus:
 - Acute midbrain syndrome
 - Acute bulbar brain syndrome
 - Upper pons stage
 - Medullary stage
- } Gerstenbrand and Lücking, 1971
} Plum and Posner, 1972
- Coma avec stabilisation des phénomènes végétatifs
 - Apallic syndrome, full stage
 - Vegetative state
- Kretschmer, 1940
Gerstenbrand, 1967
Jennett, Plum, 1972
- Coma phase sortie de l'état comateux
 - AS, remission stage
- Gerstenbrand, 1967

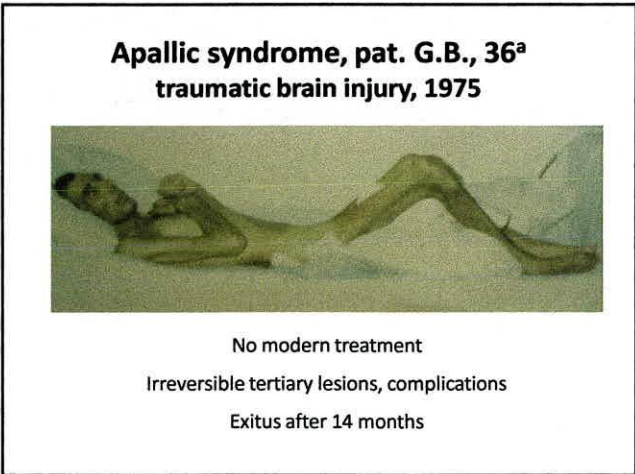
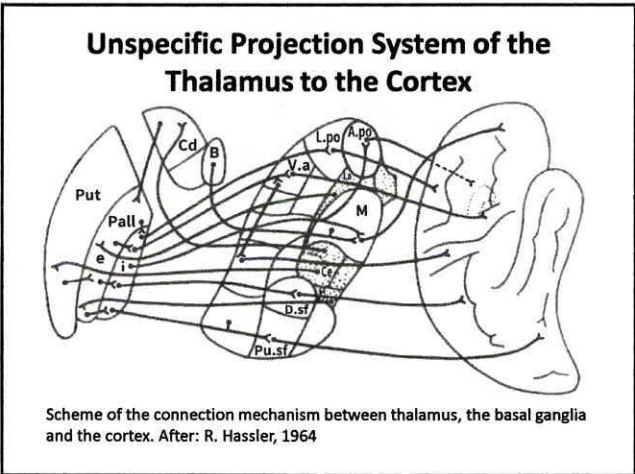
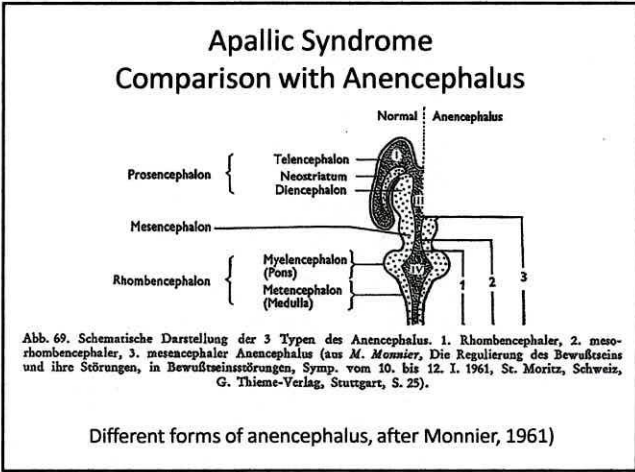
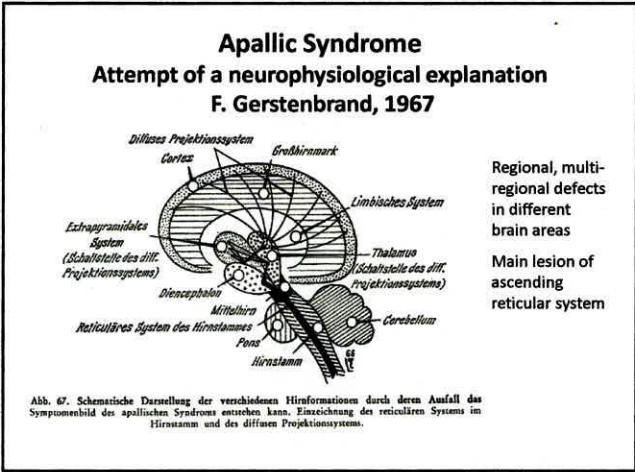


Persistent Vegetative State (PVS)

Critical aspects

- Persistent Vegetative State
 - Term as a mixture of diagnoses and prognosis
 - Only suboptimal rehabilitation possible (B. Jennet, 2002)
- Vegetative State
 - no detailed neurological description of full stage
 - No pathophysiological analysis
 - no description of initial stage, transitory stage, remission stage
 - assumed as a static condition (B. Jennet, 2002)
 - No therapeutic concept (B. Jennet, 2002)

- critics of the international community and pro life committee of catholic bishops in the US. "The term vegetative can suggest the patient is a vegetable, therefore it is subhuman and discriminatory".



Apallic syndrome, pat. E.S., 19^a
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



Pat. G.N., 39a

Full stage, traumatic apallic syndrome
Optic oral mechanism, Bulldog-reflex



Abb. 17. Vollstadium des traumatischen apallicen Syndroms. Defektstadium Übergangsstadium (Fall 1286). 1) Optischer oraler Mechanismus, 2) Optisch-oraler Mechanismus, 3) Bulldog-Lächeln, Übergang in Stage 4.

Apallic Syndrome
full stage, traumatic



Abb. 20. Vollstadium des traumatischen apallicen Syndroms (Fall 2), tonisches Greifen.
Abb. 21. Vollstadium des traumatischen apallicen Syndroms (Fall 1049), phasisches Greifen.

- Grasping reflex
 - Abb. 20: tonic
 - Abb. 21: phasic

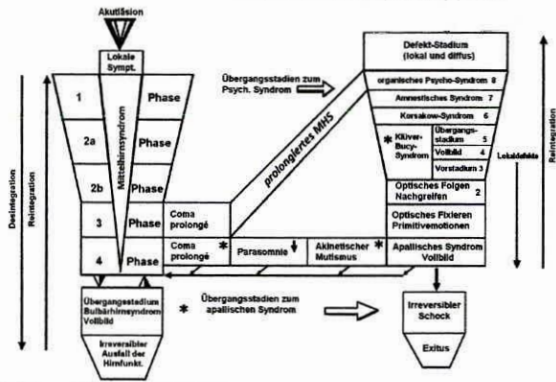
Apallic Syndrome, traumatic
full stage



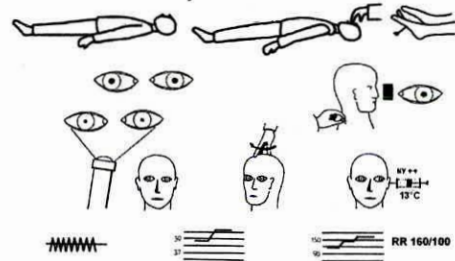
Abb. 18. Greifen mit Phasieren, tonisches (Fähigkeit nach Heidenhain, Fall 1 (DK L. 1984)). Diffuse Anomalie des Greifens, tonische Reaktion, Kompressionstendenz im Thalamus, Cerebrum präfrontales Gyrus.

L.G., 32a, full stage, died 9 months after accident.
Diffuse lesion of the white matter frontotemporal with local cystic necrosis, arterial compression necrosis in thalamus, cystic lesions periaqueductal. (Heidenhain)

Apallic Syndrome
Course after acute severe brain damages (traumatic, hypoxic, etc.)
F. Gerstenbrand, 1967, 1977, F. Gerstenbrand, E. Rimpl, 1983



Acute Midbrain Syndrome, central,
phase V

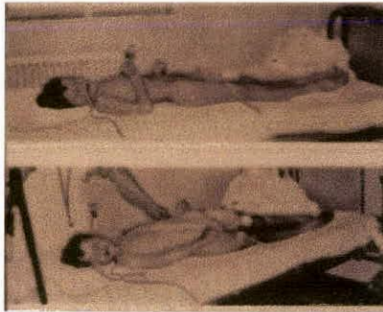


Akutes traumatisches Mittelhirnsyndrom, 4. Phase (Vollbild) Schematische Darstellung:
- Körperhaltung, Reaktion auf Schmerzreize (Orbita-Druck), Babinski'sches Zeichen.
- Pupillenweite, Reaktion auf Licht, oculo-spinaler Reflex (Knieflex am oberen Trapsirand).
- Bulbustellung, oculo-oculärer Reflex (Poppenkopf-Phänomen), vestibulo-oculärer Reflex (Kalkalibration mit Leitungswasser).
- Atmung, Temperatur, Puls, Blutdruck.

Phase V, Stretch position, disinhibition of autonomic system

Acute Secondary Midbrain Syndrome

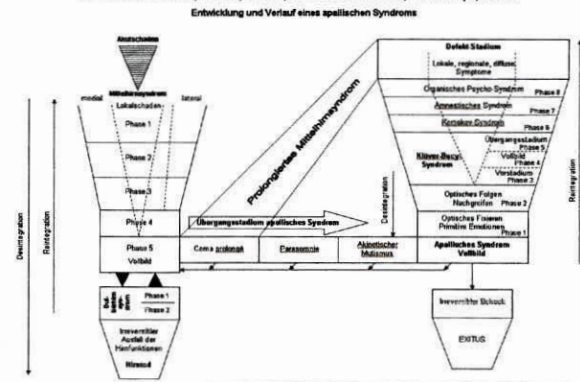
Traumatic brain injury, brain edema



Phase IV, V

Apallic Syndrome

Course after acute severe brain damages (traumatic, hypoxic, etc.)
F. Gerstenbrand, 1967, 1977, F. Gerstenbrand, E. Rimpl, 1983



STADIEN DER HIRNTAMMCHÄNDEN NACH SUPRATENTORIELLER RAUM- FORDERUNG ZENTR. HERNIATION

		MHS				BHS		
		I	II A	II B	III	IV	I	II
VIGILANTZ		SOMNOLENT	SOPOR	COMA	COMA	COMA	COMA	COMA
REAKTION	AKUSTISCHE REIZE	GERING VERZÖGERT MIT ZUWENDUNG	VERZÖGERT ODER ZUWENDUNG	FEHLEND	FEHLEND	FEHLEND	FEHLEND	FEHLEND
	SCHMERZREIZE	WICHTIG GERICHTETE ABWEHR	VERZÖGERT UNGERICHTETE ABWEHR	RESTE UNGERICHTETER ABWEHR	BRÜCK- STRECK- STELLUNG	STRECK- STRECK- SYNERGISM.	REST- STRECK- SYNERGISM.	FEHLEND
OPTOMOTORIK	STELLUNG	NORMAL	NORMAL	BEUGENHÖR DIVERGENZ	DIVERGENZ	DIVERGENZ	DIVERGENZ FOKIERT	DIVERGENZ FOKIERT
	BULBUS- BEWEGUNG	PENDELND	SCHWIMMEND	DYSKUNLIGHERT	FEHLEND	FEHLEND	FEHLEND	FEHLEND
	PUPILLENWEITE	●●	●●	●●	●●	●●	●●	●●
	LICHTREAKTION	●●	●●	●●	●●	●●	●●	●●
KÖRPER- MOTORIK	KÖRPERHALTUNG	SPONTAN- MOTORIK	MASSEN- UND WÄLZ- BEWEGUNGEN	MASSENBEWEG. ARM- STRECKBEWEG. BEINE	BEUG- STRECK- HALTUNG	STRECK- HALTUNG	REST- NACH- STRECK- HALTUNG	SCHLAFTE HALTUNG
	TONUS	NORMAL	REINE GERING ERHÖHT	BEINE ERHÖHT	ERHÖHT	STARK ERHÖHT	GERING ERHÖHT	SCHLAF
	BABINSKI PHÄNOMEN	↓	↑	↑	↑	↑	↑	—
OBIGAT	ATMUNG	—	—	—	—	—	—	—
	PULS	LEICHT ERHÖHT	NORMAL	BESCHLEUNIGT	BESCHLEU- NIGT	STARK BESCHLEU- NIGT	BESCHLEU- NIGT	VERLANGSAMT
NICHT OBIGAT	RR	NORMAL	NORMAL	NORMAL	LEICHT ERHÖHT	ERHÖHT	NORMAL	ERNIEDRIGT
	KÖRPER- TEMPERATUR	NORMAL	NORMAL	LEICHT ERHÖHT	ERHÖHT	STARK ERHÖHT	ERHÖHT	NORMAL ERNIEDRIGT

Transitory stage of apallic syndrome, development of detailed symptoms

	Akutes Mittelhirnsyndrom	Übergangsstadium		Apallisches Syndrom Vollbild
		Coma probans	Parasomnie	
Vigilanz, Coma vigile	—	—	—	—
Bewusstsein	—	—	—	—
Muskeltonus, Rigidospastizität	—	—	—	—
Position der Extremitäten	○=	○=	○=	○=
Sehnenreflexe, gesteigert	—	—	—	—
Motorische Primärschablonen oral, Greifen, spontan	—	—	—	—
Motorische Primärschablonen auslösbar durch Reize	—	—	—	—
Störung der Pupillenregulation	—	—	—	—
Oculocephaler Reflex	—	—	—	—
Vestibulooculärer Reflex	—	—	—	—
Extrapyramidale Zeichen	—	—	—	—
Vegetative Dysregulation	—	—	—	—

Apallic Syndrome after progredient, diffuse brain processes as final stage

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

- Desintegration of higher and highest brain function
Diffuse organic psychosyndrome
- Multilocular cerebral Symptoms
Aphasia, Apraxia, motoric disabilities, etc.
- Klüver-Bucy Phase
3 different stages
- Pre-apallic Phase
„dementia“, motoric primitive patterns, mass movements, decerebrate rigidity, etc.
- Apallic Syndrome, Full Stage
No remission signs

Apallic Syndrome - Remission Stages Innsbruck Remission-Scale - 1

- Phase I: Optic fixation – reduction of Coma vigile, sopor
- Phase II: Optic tracking – sleep-wake-rhythm normalized, stupor
- Phase III: Pre-Klüver-Bucy-phase – combination of primitive motor reflexes, hypersomnia – awake
- Phase IV: Klüver-Bucy-phase – typical Klüver-Bucy reflexes, obnubilation

Apallic Syndrome - Remission Stages Innsbruck Remission-Scale - 2

- Phase V: Post-Klüver-Bucy-phase – hypersomnia, communication possible
- Phase VI: Korsakov syndrome – voluntary behavior, disorientation, confusional state
- Phase VII: Amnestic phase – emotional irritaion, flat emotions
- Phase VIII: Psycho-organic syndrome – clear consciousness, aware

Symptoms of Locked-in syndrome

- No possibility to communicate with surrounding
- 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

- Disconnection of the motor system, sensory connections undisturbed

Different Types of LIS (after Bauer et al)

- Classic Locked-In syndrome
- Complete Locked-In syndrome
- Passagere Locked-In syndrome (semblance death, Scheintod)

Patient L.I.S , 45^a, female



Post traumatic etiology

Defect state

Profound differences between apallic syndrome and locked-in syndrome

- Apallic syndrome
Loss of all brain functions, reduction to the midbrain-level (coma vigile, no voluntary motor action, primitive motor patterns)
temporary or permanent

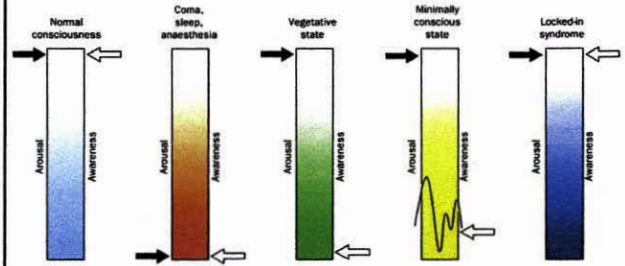
- Locked in syndrome
Loss of all motoric abilities, except rest in optomotor functions, undisturbed vigilance, full contact to the surrounding, normal body sensation
temporary or permanent

Minimally Conscious States

(Giacoia et al, 1997)

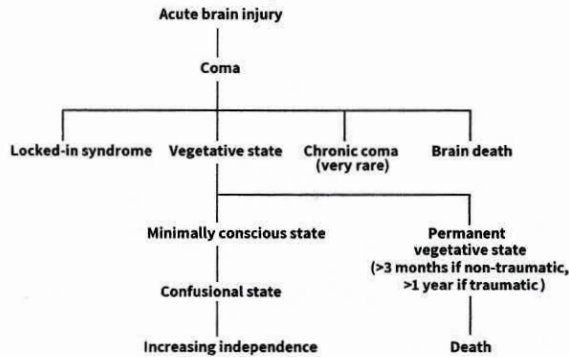
- Crude consciousness: alertness
- Phenomenal consciousness: registration of external and internal phenomena
- Access consciousness: directed attention, cognitive awareness, decision making
- Critics:
 - No detailed neurological symptomatology
 - Only phenomenological description
 - In some cases to compare with a remission phase AS/VS
 - Etiology generally open

Remission of a coma state after Laureys et al

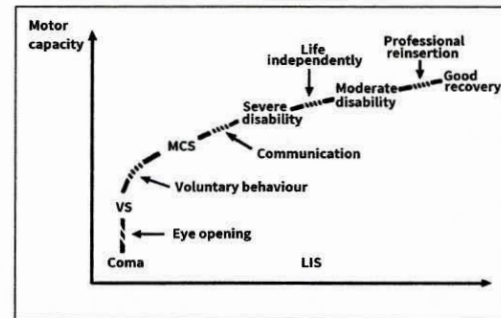


Arousal and awareness, the 2 components of consciousness in coma, vegetative state, minimally consciousness state and Locked-In syndrome

Severe acute brain injury after Laureys et al, 2004



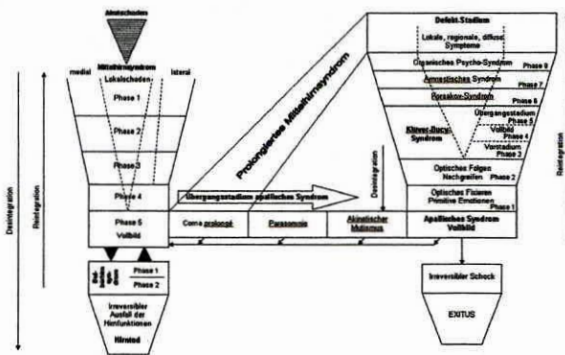
Restoration of a Coma State after Laureys et al



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

Apallic Syndrome

Course after acute severe brain damages (traumatic, hypoxic, etc.)
F. Gerstenbrand, 1967, 1977, F. Gerstenbrand, E. Rumpel, 1983
Entwicklung und Verlauf eines apallischen Syndroms



Partly „Online“ Brain Statuses

- Akinetic 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

Change ment in Consciousness

- Akinetic mutism (Cairns et al, Skultety)
 - Disturbance in the initiation of spontaneous and intentional movement
 - Awareness undisturbed
- Sopor
 - Abnormal deep sleep, not awakenable
- Stupor (Plum, Posner)
 - Deep sleep, unresponsiveness, temporarily arousable
- Hypersomnia (Jefferson)
 - Dormancy, continuously, not arousable
- Parasomnia (Facon et al.)
 - Permanent dormancy, awakening on its own after months

Offline - Online

Which structures are broken down?

- Cortical network for the different detailed brain functions
- Activation system of the cortical network (ascending reticular system)
- Functioning working system to accept and evaluate incoming stimuli as well as control of outgoing messages
- Access to the archive of memories and ability to add new experiences

Offline – Online

„Main Operating System“

- Ascending reticular system
 - Functioning
 - unimpaired
 - Activation with different methods
 - Stimulation with all incoming sensory stimuli
 - Optic and acoustic stimulation, etc.
 - Stimulation of proprioceptive system
 - Medication
- Functional, biochemical, physical activation
 - Operating like a “joy stick”
- “Switches” unknown

The 42nd International Danube Neurology Symposium (by Professor Vida Demarin)

Date: 21-23 October, 2010

Place: The Regent Esplanade Hotel, Zagreb, Croatia

Wednesday, October 20, 2010

19:00 Opening Ceremony and welcome reception

Thursday, October 21, 2010

Main Theme: Stroke

(Convenors: Vida Demarin, Zagreb, Ana Czlonkowska, Warsaw)

09:00 - 09:30	Ana Czlonkowska:	TIA as an Emergency
09:30 - 10:00	Natan Bomstein:	Management of Hypertension and Hyperglycemia in Acute Ischemic Stroke
10:00 - 10:30	Nadežda Sternić:	Diabetes and Stroke
10:30 - 11:00	Break	
11:00 - 11:30	Kurt Niederkorn:	Neurosonology in Acute Stroke
11:30 - 12:00	Vida Demarin:	Recent Concept of Stroke Prevention
12:30 - 13:00	Dafin Muresanu:	The Impact of Co-morbidities and Neuroprotective Treatments in Stroke Recovery

13:00 Lunch symposium: Approach to aging brain
(Convenors: Danilo Hodoba, Zagreb, Zvezdan Pirtošek, Ljubljana)

15:00 - 17:00	Workshop:	Diagnosis of Brain Death
16:00 - 17:00	Franz Gerstenbrand:	The off-line Brain - is there such a thing?

15:00 - 17:00	Teaching course:	Fabry Disease and Enzyme Replacement Therapy in Neurology
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(Convenors: Vida Demarin, Zagreb, Vanja Bašić Kes, Zagreb)

15:00 - 17:00 Society for the Study of Neuroprotection and Neuroplasticity (SSIMIM) Panel
(Convenor: Dafin Muresanu, Cluj)

17:00 - 18:00	Franz Gerstenbrand:	Modern neurology and the Hippocratic principles
17:00 - 19:00	Posters	

Friday, October 22, 2010

Main Theme: Movement Disorders - Non-Motor Symptoms in Parkinson's Disease
(Convenors: Maja Relja, Zagreb, Zvezdan Pirtošek, Ljubljana)

09:00 - 09:30	Werner Poewe:	Continuous dopaminergic stimulation in Parkinson's disease
09:30 - 10:00	Maja Relja:	Non-motor symptoms in Parkinson's disease
10:00 - 10:30	Vladimir Kostić:	Depression in Parkinson's disease
10:30 - 11:00	Break	
11:00 - 11:30	Zvezdan Pirtošek:	Cognition in Parkinson's disease
11:30 - 12:00	David Vodušek:	Urogenital symptoms in differential diagnosis of Parkinsonism

Joint Meeting of Danube Society for Neurological Sciences and Continuing Education with Central and Eastern European Stroke Society (CEESS) and Croatian Stroke Society: Stroke Management

09:00 - 09:30 Ana Czlonkowska: Thrombolysis in the Region
09:30 - 10:00 Ljiljana Bumbaširević: Stroke Unit - Secondary Prevention Center
10:00 - 10:30 Vida Demarin: Carotid Disease
10:30 - 11:00 Bojana Žvan: Management of subarachnoidal hemorrhage
11:00 - 11:30 Osman Sinanović: Post-stroke aphasia

09:00 - 17:00 Cochrane European Association Young Neurologists and Trainees (EAYNT) Workshop

15:00 - 17:00 Experimental Pain and Therapy
(Convenors: Claudia Sommer, Wuerzburg, Zdravko Lacković, Zagreb)

Claudia Sommer: Therapy of neuropathic pain
Lidia Bach-Rojecky: Experimental model of pain
Zdravko Lacković: Central effects of botulinum toxin-type A
Alfredo Berardelli: The impact of botulinum toxin treatment on cortical excitability
Maja Relja: Botulinum toxin in migraine treatment

17:00 - 19:00 Teaching Course: Dystonia: Diagnosis and Treatment

17:00 - 18:00 Franz Gerstenbrand: Space Neurology and the use of astronaut/cosmonauts equipments in neurorehabilitation

17:00 - 19:00 Posters

Saturday, October 23, 2010

Main Theme: Headache and Pain
(Convenors: Laszlo Vecsei, Szeged, Zdravko Lacković, Zagreb)

09:00 - 09:30 Laszlo Vecsei: The Role of Kynurenate Derivate in Nociception
09:30 - 10:00 Zdravko Lacković: Experimental Model of Migraine
10:30 - 11:00 Vanja Bašić Kes: Migraine and Stroke Connection
11:00 - 11:30 Ivo Lušić: Central Post-stroke Pain
12:00 Closing ceremony

All participants are most welcome to participate in all social and scientific activities

Deadline for abstract submission: June 1, 2010.

Congress venue: REGENT ESPLANADE HOTEL

Mihanovićeveva 1

10000 Zagreb, Croatia

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All abstracts will be available on CD and via CROSB, editor Vida Demarin.

ZAGREB 2010



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EVIDENCE-BASED NEUROLOGY

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OF THE CROATIAN MEDICAL ASSOCIATION AND THE CROATIAN STROKE
SOCIETY

10th CONGRESS OF EUROPEAN SOCIETY FOR CLINICAL NEUROPHARMACOLOGY

JOINT MEETING OF CENTRAL EASTERN EUROPEAN STROKE SOCIETY



Gerstenbrand, Franz ; Binder, Heinrich; Golaszewski, Stefan;

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Zagreb, 2010. str. CD-CD (plenarno, nije recenziran, sažetak, znanstveni)

Naslov

THE OFF-LINE BRAIN - IS THERE SUCH A THING?

Title**Autori**

Gerstenbrand, Franz ; Binder, Heinrich; Golaszewski, Stefan;

Autors**Vrsta, podvrsta i kategorija rada**

Sažeci sa skupova, sažetak, znanstveni

Type, subspecies and category of work

Summary of meetings, abstracts, scientific

Izvornik

Abstracts: *The 42nd International Danube Neurology Symposium October 21 - 23, 2010, Zagreb CD* /

Vida Demarin - Zagreb, 2010. CD-CD

Original**Skup**

The 42nd International Danube Neurology Symposium

October 21 - 23, 2010, Zagreb

Event**Mjesto i datum**

Zagreb, Hrvatska, 21.-23. listopada 2010

Location and date**Vrsta sudjelovanja**

Plenarno

Kind of participation

Plenary

Vrsta recenzije

Nije recenziran

Type of review

No review

Ključne riječi

coma vigilie; apallic syndrome/vegetative state;
brain death syndrome; locked-in syndrome;

Keywords

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Sažetak**Abstract**

Coma vigilie is a special form of coma, the leading symptom of the apallic syndrome/vegetative state. The patient is conscious, but without higher and highest brain functions and shows more or less uniform neurological symptoms.