Apallic Syndrome – New Developments

F.Gerstenbrand[1), 2)], S.M. Golaszewski [1),3)], Ch. Kurzmann[1)],

[1)] Karl Landsteiner Institute for Neurorehabilitation and Space Neurology
[2)] Department of Neurology, University of Innsbruck
[3)] Department for Neurology, Christian Doppler-Klinik Salzburg

Danube Teaching Course
Kazimierz Dolny
September 13th - 15th, 2012

Epidemiology of Apallic Syndrome Divergences in statistic evaluation

Prevalence 1.9/100000 pop./year in Austria (160 pat.)

Prevalence 1,7/100000 pop./year in Germany (1.500 pat.)

Prevalence in Italy and Belgium 0.9 – 2.0/100000 pop./year

Incidence USA 15000-35000 pat./year

Incidence Great Britain 1500 pat./year

Incidence France 1000-1200 pat./year (post-traumatic)

Incidence Japan 10000-17000 pat./year

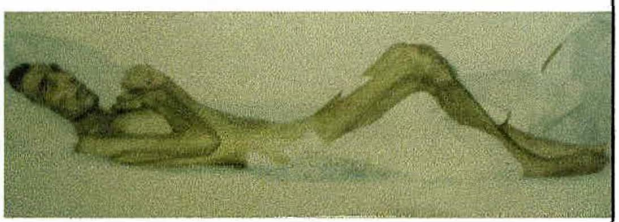
Etiology of Apallic Syndrome

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*

Symptoms of Apallic Syndrome - full stage

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

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



No modern treatment

Irreversible tertiary lesions and complications

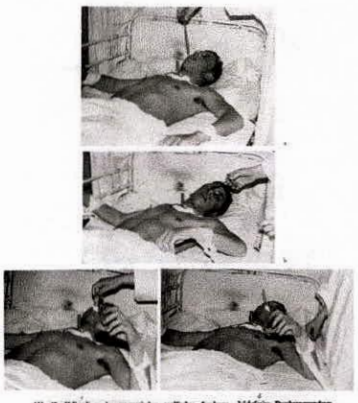
Exitus after 14 months

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

Modern treatment program in special center for apallic syndrome patients, Innsbruck

No tertiary lesions, minimal complications

Remission after 5 months to minimal defect state



Pat. G.N., 39a

- Full stage, traumatic apallic syndrome
- Optic induced oral crasping refelexes, Bulldogg-reflex

Apallic Syndrome
Full stage, traumatic etiology

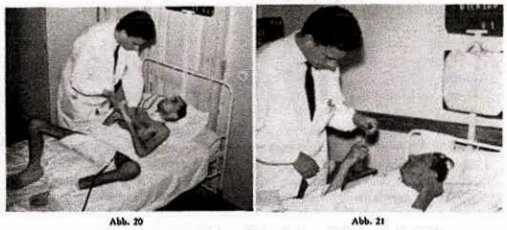


Abb. 20 Abb. 21

Abb. 20. Vollstadium des traumatischen apallischen Syndroms (Fall 2), tonisches Greifen.
Abb. 21. Vollstadium des traumatischen apallischen Syndroms (Fall 10/40), phasisches Greifen.

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

Traumatic Apallic Syndrome, full stage

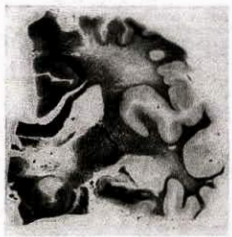


Abb. 18. Großhirn mit Hirnstamm, Frontalschnitt (Färbung nach *Heidenhain*), Fall 1 (K. L. 107/43). Diffuse fronto-temporale Markläsion, cystische Herdnekrosen, Konsistenzminderung im Thalamus. Cysten im periaquäduktalen Gebiet.

L.G., 32a, full stage, died 9 months after accident,
Diffuse lesion of the white matter fronto-temporal with local cystic necrosis, necrosis in thalamus, cystic lesions peri-aquaeductal.
(Heidenhain staining)

Pathophysiology of apallic syndrome (AS)

- Pallium, Latin term for old Greek overcoat Signalizing a functional disturbance
- Disconnection of cortical function to brain stem centers
- Anatomical basis:
  - Lesion of the ascending reticular system
  - depending to the course and to the defect:
    - Discrete damage of cortical and sub-cortical regions (local, regional)
    - Severe damage of the cortex (laminar lesion) and/or diffuse or local damage of sub-cortical areas

Location of lesions causing apallic syndrome
F. Gerstenbrand, 1967

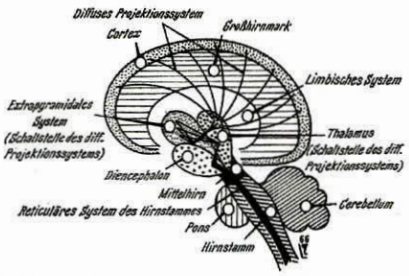


Abb. 67. Schematische Darstellung der verschiedenen Hirnformationen durch deren Ausfall das Symptomenbild des apallischen Syndroms entstehen kann. Einzeichnung des reticulären Systems im Hirnstamm und des diffusen Projektionssystems.

Apallic Syndrome
Comparison with Anencephalus

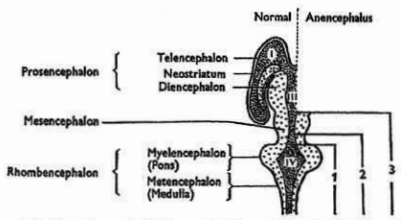
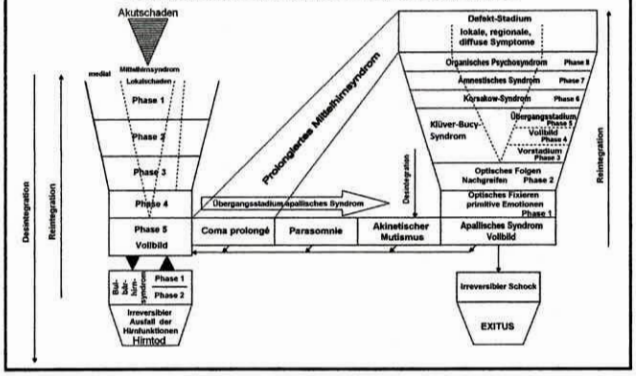


Abb. 69. Schematische Darstellung der 3 Typen des Anencephalus. 1. Rhombencephaler, 2. mesorhombencephaler, 3. mesencephaler Anencephalus (aus *M. Monnier*, Die Regulierung des Bewußtseins und ihre Störungen, in Bewußtseinsstörungen, Symp. vom 10. bis 12. I. 1961, St. Moritz, Schweiz, G. Thieme-Verlag, Stuttgart, S. 25).

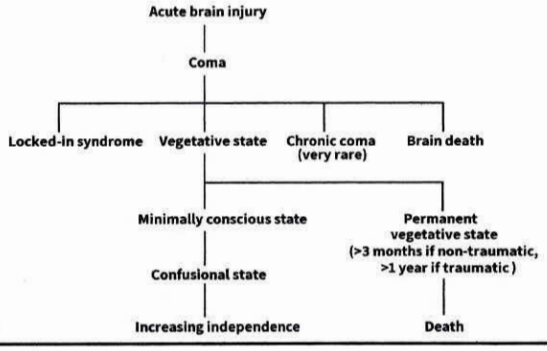
Different forms of anencephalus, after Monnier, 1961)

The course of apallic syndrome after acute brain damage

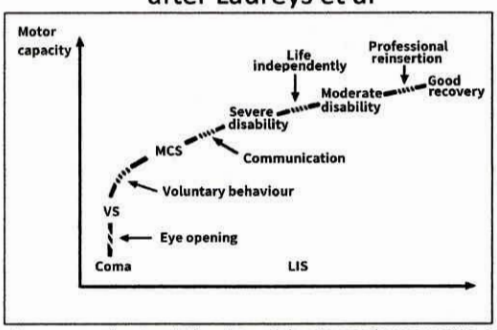
F. Gerstenbrand, 1967, 1977, F. Gerstenbrand, E. Rumpl, 1983



Clinical Course
Severe acute brain injury
Laureys et al., 2004



Restauration of a Coma State after Laureys et al



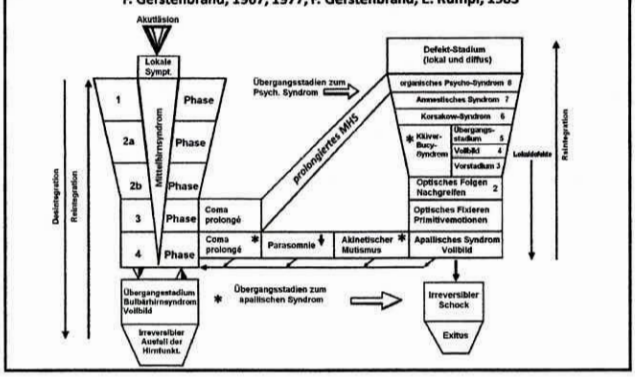
Acute coma, passing, vegetative state, minimally consciousness state, good recovery

Etiology of apallic syndrome

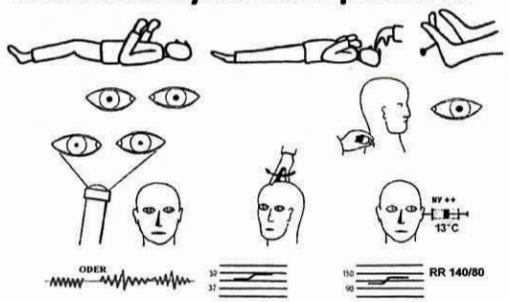
1. Acute, severe brain injury
   - TBI, encephalitis, hypoxia, malignant stroke etc.
     - Possibility of remission
2. Progredient, diffuse brain processes
   - CJD, M.Alzheimer, M.Pick, Chorea Huntington etc.
   - Final stage
     - Remission not possible
3. Intoxication
   - 3a Acute form
     - Exogenous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

The course of apallic syndrome after acute brain damage

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



Midbrain syndrome phase III



Akutes traumatisches Mittelhirnsyndrom, 3. Phase. Schematische Darstellung:
- Körperhaltung, Reaktion auf Schmerzreize (Orbita-Druck), Babinski'sches Zeichen.
- Pupillenweite, Reaktion auf Licht, cilio-spinaler Reflex (Kneifen am oberen Trapezlusrand).
- Bulbusstellung, oculo-cephaler Reflex (Puppenkopf-Phänomen), vestibulo-oculärer Reflex (Kaltkalorisation mit Leitungswasser).
- Atmung, Temperatur, Puls, Blutdruck.

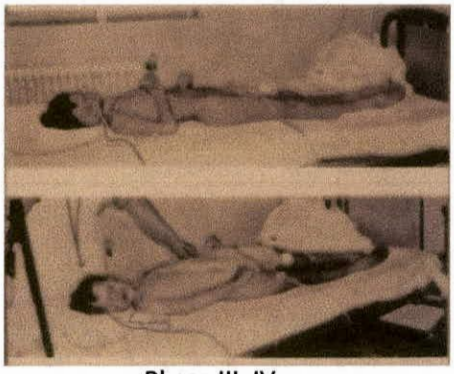
Phase III, Stretch-Flex position, disinhibition of autonomic system

Midbrain syndrome, phase IV

- Coma
- Threat reflex absent
- Divergent position of bulbi
- Pupils reduced reaction to light
- Oculo-cephalic reflex blocked
- Vestibulo-ocular reflexes dissociated
- Stretch position: extremities, trunk
- Increased muscle tone, pyramidal signs
- Respiration – machine like rhythm
- Hyperthermia, tachycardia, increased blood pressure

Acute secondary midbrain syndrome

Traumatic brain injury, brain edema



Phase III, IV

STADIEN DER HIRNSTAMMSCHÄDEN NACH SUPRATENTORIELLER RAUMFORDERUNG ZENTR. HERNIATION		MHS					BHS	
		I	II A	II B	III	IV	I	II
VIGILITÄT		SOMNOLENZ	SOPOR	COMA	COMA	COMA	COMA	COMA
REAKTION	AKUSTISCHE REIZE	GERING VERZÖGERT MIT ZUWENDUNG	VERZÖGERT OHNE ZUWENDUNG	FEHLEND	FEHLEND	FEHLEND	FEHLEND	FEHLEND
	SCHMERZREIZE	PROMPT GERICHTETE ABWEHR	VERZÖGERT UNGERICHTETE ABWEHR	RESTE UNGERICHTETER ABWEHR	BEUGE-STRECK-STELLUNG	STRECK-SYNERGISMEN	REST-STRECK-SYNERGISM.	FEHLEND
OPTOMOTORIK	-STELLUNG BULBUS -BEWEGUNG	NORMAL PENDELND	NORMAL SCHWIMMEND	BEGINNENDE DIVERGENZ DYSKUNUGIERT	DIVERGENZ FEHLEND	DIVERGENZ FEHLEND	DIVERGENZ FIXIERT FEHLEND	DIVERGENZ FIXIERT FEHLEND
	PUPILLENWEITE							
	LICHTREAKTION							
KÖRPER-MOTORIK	KÖRPERHALTUNG							
	SPONTAN-MOTORIK	MASSEN- UND WÄLZ-BEWEGUNGEN	MASSENBEWEG. ARME STRECKBEWEG. BEINE	MASSENBEWEG. ARME STRECKBEWEG. BEINE	BEUGE-STRECK-HALTUNG	STRECK-HALTUNG	REST-; NACH-STRECK-HALTUNG	SCHLAFFE HALTUNG
	TONUS	NORMAL	BEINE GERING ERHÖHT	BEINE ERHÖHT	ERHÖHT	STARK ERHÖHT	GERING ERHÖHT	SCHLAFF
	BABINSKI PHÄNOMEN	↓ ↓	↑ ↓	↑ ↓	↑ ↑	↑ ↑	↑ ↑	——
OBLIGAT	ATMUNG							——
VEGETATIV	PULS	LEICHT ERHÖHT	NORMAL	BESCHLEUNIGT	BESCHLEUNIGT	STARK BESCHLEUNIGT	BESCHLEUNIGT	VERLANGSAMT
NICHT OBLIGAT	RR	NORMAL	NORMAL	NORMAL	LEICHT ERHÖHT	ERHÖHT	NORMAL	ERNIEDRIGT
	KÖRPER-TEMPERATUR	NORMAL	NORMAL	LEICHT ERHÖHT	ERHÖHT	STARK ERHÖHT	ERHÖHT	NORMAL ERNIEDRIGT

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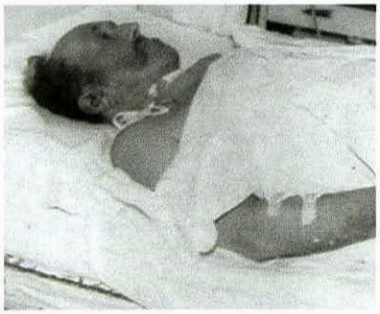
   Exogenous (Minamata disease etc.)
   Endogenous (hepatic, thyreotoxic etc.)
   Partial remission possible

Apallic Syndrome after progredient, diffuse brain processes to a final stage

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

- Desintegration of highest and higher brain functions
  (Diffuse organic psychosyndrome)
- Multilocular cerebral Symptoms
  Aphasia, Apraxia, motor symptoms, etc.
- Klüver-Bucy Phase
  3 different stages
- Preapallic Phase
  „severe dementia", primitive motor patterns, massmovements, decorticate rigidity, etc.
- Apallic Syndrome, Full Stage
  No remission signs

Apallic syndrome, full stage Alzheimer disease, end phase



Terminology
Recent Discussion

- Apallic Syndrome
- Vegetative State
- Wachkoma – Coma Vigile
- Unresponsive Wakefulness
- Minimally Conscious State

Apallic Syndrome
Synonyma in literature

- Rosenblath: Über einen bemerkenswerten Fall von Hirnerschütterung, 1899
- Hermann: Livedo racemosa, 1937
- Kretschmer: Das apallische Syndrom, 1940
- Cairns et al.: acinetic mutism, 1941
- Duensing: Anoetischer Symptomenkomplex, 1949
- Jefferson: Parasomnie, 1952
- Ajuriaguerra et al.: Luzider Stupor, 1954
- Strich: Severe dementia following head injury, 1956
- Fischgold und Mathis: Stupeur hypertonique postcomateuse, 1959
- Sutter et al.: Catatonie posttraumatique, 1959
- Jellinger et al.: Protrahierte Form der posttraumatischen Encephalopathie, 1963
- Osetowska: Leucoéncephalopathie oedémateuse posttraumatique, 1964
- Gruner: démence progressive avec cachexie, 1965
- Gerstenbrand: Apallisches Syndrom, 1967
- Jenett und Plum: Persistent vegetative state (PVS), 1972
- Giacino und Zasler: Minimally response state, 1995

Persistent Vegetative State (PVS)
Identical symptoms to Apallic Syndrome - critical aspects

- Vegetative State – Persistent Vegetative State: Term mixes diagnoses and prognoses, only sub-optimal rehabilitation is possible (B. Jennet, 2002)
- Vegetative State, a detailled neurological description and analysis is not existing, no description of initial stage, transitory stage, full stage and remission stage)
- Vegetative State: assumed as a static condition (B. Jennet, 2002)
- Vegetative State: no therapeutical concept (B. Jennet, 2002)
- Vegetative State: critics of international community and pro life committee of catholic bishops in the USA. The word vegetative can suggest the patient is a vegetable, therefore subhuman and discriminatory.
- Critics of English and American experts to use the term „wakeful unconscious state"(1995), „unresponsible wakefulness"(2012).

Wachkoma - Coma Vigile
(Donis, 2011)

- Spontaneous or reflectoric opening of the eyes
- Absence of self recognition
- Absence of recognition of surrounding
- Absence of meaningful reactions
- No communication to surrounding
- No optic fixation, no optic tracking
- Persisting of sleep-wake rhythm
- Neurological deficits
  - Optomotoric disturbancies
  - Flexed-strech position, rigido spasticity
  - Motoric primitive patterns (oral, crasping etc.)
  - Brain stem reflexis present
  - Initial stage (acute midbrain, bulbar brain syndrome)

Unresponsive Wakefulness (UWS)

K.v.Wild, St.Laureys, G.Dolce, 2010

- Eyes open, spontaneous or after stimulation
- sleep-wake rhythm present
- No reaction to external stimulation
- Automatic motor reflexes
- Spontaneous respiration
- Persistance of thermoregulation and digestion

Minimally Conscious States

(Giacino et al, 1997)

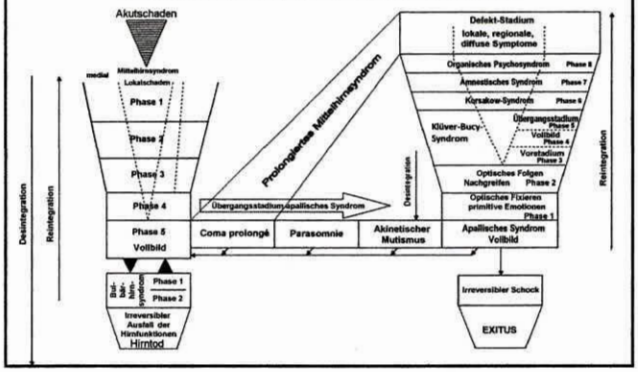
No identical symptoms to Apallic Syndrome

- 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
  - To compare with remission course of AS
  - Etiology generally open

The course of apallic syndrome after acute brain damage

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



Apallic Syndrome - Remission Stages
Innsbruck Remission-Scale - 1

- Phase I: Optic fixation – reduction of Coma vigile, sopor
- Phase II: Optic tracking – sleep-wake-rhythm nomalizing, stupor
- Phase III: Pre-Klüver-Bucy-Phase – combination in the primitive motor reflexes, hypersomnia – wakeful
- Phase IV: Klüver-Bucy-Phase – Klüver-Bucy reflexes, obnubilation

Apallic Syndrome Development

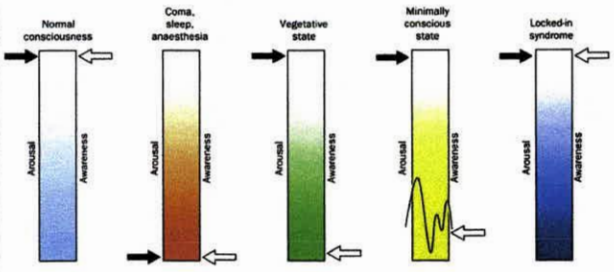
Full-stage of traumatic apallic syndrome

Early remission stage

Apallic Syndrome - Remission Stages
Innsbruck Remission-Scale - 2

- Phase V: Post-Klüver-Bucy-Phase – hypersomnia, first voluntary motor skills, communication possible
- Phase VI: Korsakov syndrome – voluntary behavior, disorientation, confusion state
- Phase VII: Amnestic phase – emotional irritation, flat emotions
- Phase VIII: Psycho-organic syndrome – normal consciousness, awake
  local lesions with neurological symptoms

Remission of a Coma State (Laureys et al)



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

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

- Alpha-EEG

Etiology of Locked-In Syndrome Lesion in Pons

- Pons infarction caused by basilar thrombosis
- Hemorrhage
- Encephalitis
- Tumor
- Traumatic lesion

- Disconnection of the motor system, sensory connections undisturbed

Different Forms of Locked-In Syndrome (Bauer et al, 1979)

- According to neurological symptoms and course
  - Classical Locked-In syndrome
  - Incomplete Locked-In syndrome
  - Total Locked-In syndrome
- According to time course
  - Chronic Locked-In syndrome
  - Transient Locked-In syndrome

Locked-In Syndrome, female, 45a

Traumatic etiology

Passager Apallic Syndrome

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), unresponsive wakefulness
  temporary or permanent

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

Diagnosis of Apallic Syndrome Differentialdiagnosis Locked In Syndrome

- Bedside testing, neurological examination, (Coma Recovery Scale – revised - CRS-R)
- Functional Magnetic Resonance (fMRI)
  - Verbal paradigm:
    - Semantic oddball paradigm (SOP): acoustic presentation of meaningful versus non meaningful sentences in an fMRI event related oddbal paradigm
    - Own name paradigm (ONP): acoustic presentation of the patient's own name versus not the own name with a familiar voice in an fMRI event related oddbal paradigm
  - Vibrostimulation paradigm

Functional MRI verbal paradigms:

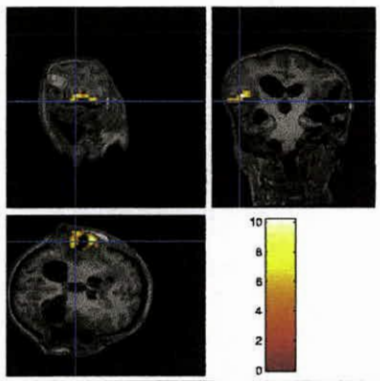
- **S > R: sentences vs rest**
- **M > NM: meaningful vs non meaningful sentences**
- **O > R: own name vs rest**
- **O > NO: own name vs not own name**

Semantic Oddball paradigm (meaningful versus non-meaningful sentences)

e.g. The sun is hot

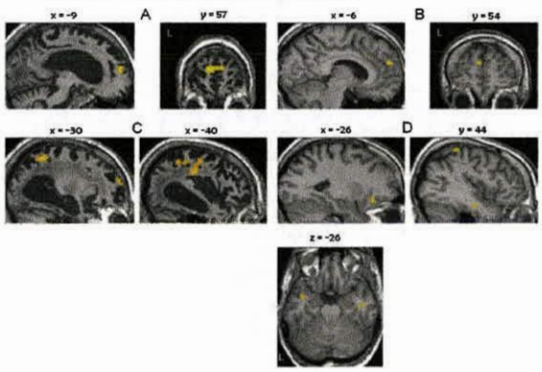
e.g. With the ears one can speak

Apallic Syndrome fMRI, verbal stimulation, Bold – effect



Apallic Syndrome, fMRI, verbal stimulation Bold – effect:

increased activity, language processing own name > foreign name (Wernicke, Broca area) (Staffen et. al, 2006)



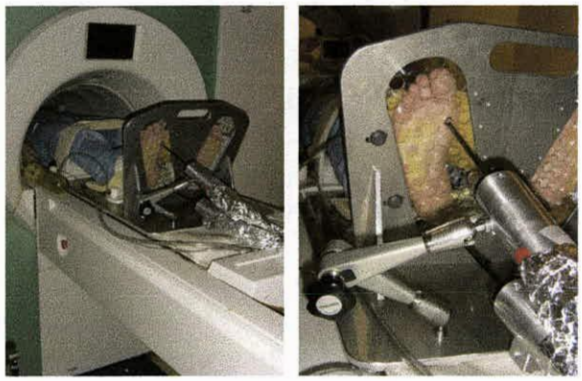
Summary

fMRI in diagnosis of Apallic Syndrome, verbal stimulation

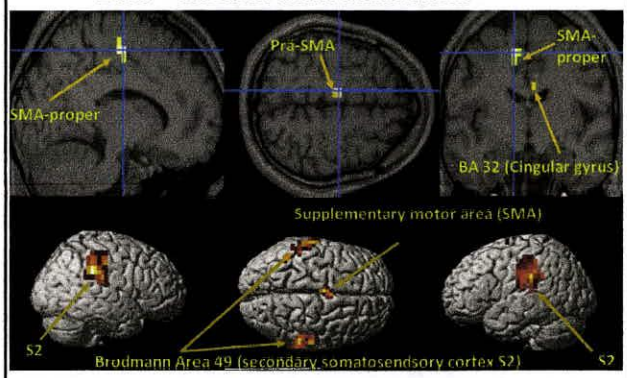
- Apallic Syndrome: fMRI shows specific activity in language regions and regions of self-awareness. Patients are able for the processing of language, memory and self-referential stimuli at a higher cortical level.
- Evidence of perception in language and self-referential stimuli in patients with Apallic Syndrome, important for individual planning of neurorehabilitation program, for the relatives, caregivers and therapists.
- In future: prognostic decisions may be possible.

Effect of vibratory stimulation in differernt brain areas:

- Vibratory stimulation of the hand palm and the sole of the foot revealed robust contralateral activation within the primary sensorimotor cortex (SM1), bilateral activation within the secondary somatosensory cortex (S2, Brodmann Area 40), bilateral within the supplementary motor area (SMA, BA 6) and ipsilateral within the cingulate gyrus (BA 32).
- Passive sensory stimulation by a vibratory stimulus to the hand palm and the sole of the foot leads to activation within the whole sensorimotor cortex like in active motor paradigms such as finger-to-thumb or foot tapping
- This holds promise for the vibratory stimulation as an alternative to active motor paradigms in neurological patients with severe motor deficits to study sensorimotor cortex functions in patients with brain pathology or pathology of afferent pathways for functional diagnosis, prognosis and monitoring of rehabilitation.

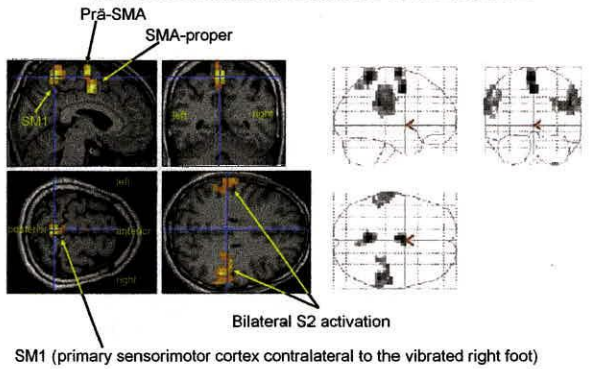


Vibration of the sole of the foot
Bold – effect in fMRI evaluation:



Vibration of the sole of the foot
fMRI evaluation: Bold – effect

brain activation within SMA/bilateral Area 40/ contralateral SM1



Locked-In Plus Syndrome

Symptoms of disturbances in the A. basilaris circulatory

- No possibility to communicate with surrounding except for blinking morse code
- Consciousness and perception mostly maintained
- Total paralysis of all extremities, trunk, neck and motor brain nerves
- Eye opening and vertical eye movements possible
- Sleep-/ awake rhythm continued
- Spontaneous respiration possible

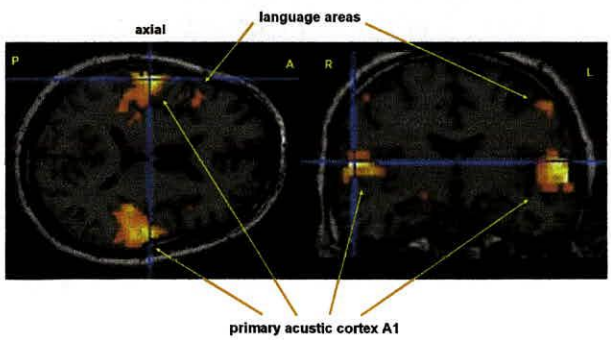
- **Additional Symptoms:**
  - Hypersomnia, parasomnia
  - Acinetic mutism
  - Thalamic symptoms
  - Optomotoric disturbances

F. Gerstenbrand, S.M.Golaszewski, G.Bauer, E.Trinka, 2011

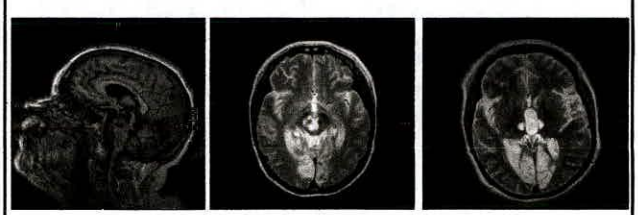
Different Forms of Disturbances in Consciousness

- 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

Locked-In-Syndrome plus, 44 y. old man, Basilar thrombosis
3 years ago, misdiagnosed as Apallic Syndrome
additional Symptoms: Hypersomnie, Parasomnia, Verbal stimulation



Locked-In-Syndrome plus, 29 year old female, additional Symptoms:
Acinetic Mutism, Hypersomnia, thalamic Symptoms



Severe local structural lesion in Basilar irrigation territory;
total LIS with accentuated defect symptoms of right thalamic lesion,
symptoms of hypersomnia, signs of an acinetic mutism

Misdiagnosed as Apallic Syndrome

Special Treatment Program in Apallic Syndrome

- Activation of the proprioceptive system
  logomatics, cosmonaut trousers etc.
- Hyperbaric Oxygenation Treatment
- Surgical interventions of complications
  (joint contractions, hydrocephalus oclusus)
- Operation of periarticular ossification
- Deep brain stimulation of thalamic intralaminar nuclei

HBOT – program Apallic Syndrome

- Start
  - in full stage
    → as early as possible
  - in remission stage with temporary interruption state II-III
    → after 3 months
  - in later remission states and in defect stage without sufficient result of running rehabilitation
    → in any case
- Continuation of classic treatment and consequent neurorehabilitation program

Apallic Syndrome, HBOT - program

remission stage

MP, 23ª, male, AS, traumatic
remission stage IV
HBOT: 1997 – 1999: 208 sessions,
1.5 – 1.75 ATA
HBOT: since April 2002: 600 sessions

Additional treatment: physiotherapy

Most light defect symptoms as spasticity, pseudo-bulbar symptoms, emotional irritability, frontal lobe symptoms
Full ability for self decisions

GCS: 6; Final GCS: normal

Apallic Syndrome, HBOT - program

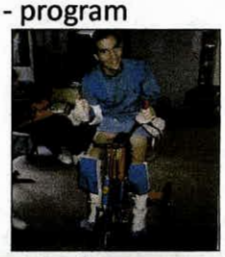
remission stage

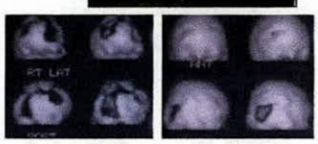
MJ, 31ª, male, AS,
CO-intoxication, suicide,
remission state II-III during 12 years.

HBOT: 60 sessions
HBO home treatment: 350 sessions
1,5 ATA

Additional:
physio-therapy
logopedics

Marked improvement
minimal cognitive defects,
spastic signs





before HBO after HBO

Apallic Syndrome, HBOT - program

remission stage

SPECT:

Marked improvement of perfusion

JN, 21ª, male, AS, traumatic
remission stage II-III
HBOT: 64 sessions
1.5 – 1.75 ATA

Additional treatment:
physio therapy

Significant improvement
Light defect symptoms: cerebellar, spastic symptoms, speech disturbances (pseudo-bulbar), cognitive deficits

Apallic Syndrome and HBO-treatment

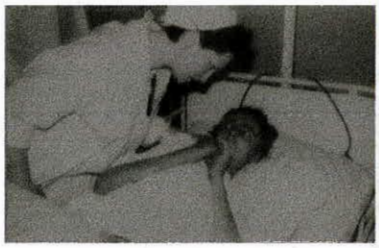
Full stage: reorganization of cortical connections to brain stem centers supported by the activation of reticular system

Remission stage: Revitalization of the cortical network (idling neurons)

Revitalization of damaged axons

Defect stage: Reactivation of dendrite system

Apallic Syndrome **Development**



Late remission stage

Full recovery

Terri Schiavo (USA)

Apallic Syndrome/vegetative state, remission state II-III, contact with the surroundings

- Emotional reaction
- Optic fixation to her mother
- Turn towards
- Emotional contact reaction
- Well-balanced body state
- Vegetative system regulated
- No artificial respiration
- Nutrition by PEG

End of life decision by court, withdrawal of liquid and nutrition.

Apallic Syndrome – sindrome apallico (traumatic), Salvatore C., 38[a]

- Traumatic brain injury, August 2003
- late onset of remission
- Defect state with neurological and orthopedic deficits

In full stage patient heard noises of the surroundings, perceived pains and physical contact. Deep desperation.

Successful rehabilitation after AS, traumatic, Fred A., 39[a]

- Car accident 1995 with 30 years
- Apallic syndrome in full stage in a special center for apallic patients over 6 months
- Remission phase over 2 years
- Treated in special rehabilitation center for apallic patients
- Continued rehabilitation with stepwise improvement
- Full integrated in family life, father of a 3 years old daughter
- Only partial handicapped
- Strict aim to build up a normal professional condition

International Danube Symposium for Neurological Sciences and Continuing Education

in collaboration with

Lublin Branch of Polish Neurological Society

Department of Neurology
Medical University of Lublin

VIII MIĘDZYNARODOWE WARSZTATY SZKOLENIOWO-NAUKOWE

STWARDNIENIE ROZSIANE, PADACZKA, BÓLE I ZAWROTY GŁOWY, NEUROLOGIA DZIECIĘCA

8TH INTERNATIONAL TEACHING COURSE

MULTIPLE SCLEROSIS, EPILEPSY, HEADACHE AND VERTIGO, CHILD NEUROLOGY

September 13-14, 2012

Kazimierz Dolny, Poland

PROGRAM
FINAL PROGRAMME

PROGRAM NAUKOWY / SCIENTIFIC PROGRAMME

Czwartek, 13 września 2012 / Thursday, September 13, 2012

SALA GŁÓWNA B-D / Main Hall B-D

9.00 – 10.30 Danube Lectures
Przewodniczący / Chairperson: Z. Stelmasiak
A. Korczyn – *Cognitive impairment in Parkinson Disease*
J. Chapman – *Is Multiple Sclerosis a disease or a syndrome with heterogeneous etiologies?*
B. Machaliński, M. Gołąb-Janowska, B. Baumert, P. Nowacki – *Adiuwantowa terapia komórkowa stwardnienia zanikowego bocznego – mit czy nadzieja? / Adjuvant cell therapy of amyotrophic lateral sclerosis – myth or hope?*

10.30 – 11.00 Przerwa na kawę / Coffee-break

11.00 – 12.30 Stwardnienie rozsiane (Sesja I) / Multiple sclerosis (Session 1)
Przewodniczący/Chairpersons: K. Selmaj, H. Bartosik-Psujek
K. Selmaj – *Korzyści i ryzyko terapii SM / Benefit and risk of MS therapy*
H. Bartosik-Psujek – *Wytyczne nowego programu terapeutycznego SM / Guidlines of MS new therapeutic program*
K. Rejdak – *Optymalizacja leczenia SM w aspekcie przeciwciał neutralizujących / Optimalizing of MS therapy – neutralizing antibody aspect*

12.30 – 13.30 Przerwa na posiłek / Lunch-break

13.30 – 14.50 Stwardnienie rozsiane II / Multiple sclerosis (Session 2)
Przewodniczący/Chairpersons: J. Kotowicz, Z. Maciejek
Z. Maciejek – *Terapia długotermiowa w SM / Long term therapy in MS*
J. Kotowicz – *Komórki macierzyste w leczeniu SM / Stem cells in the treatment of MS*
K. Mitosek-Szewczyk – *Bóle głowy a SM / Headaches and MS*

14.50 – 15.10 Przerwa na kawę / Coffee-break

15.10 – 17.30 Bóle i zawroty głowy / Headaches and vertigo
Przewodniczący / Chairpersons: A. Prusiński, K. Mitosek-Szewczyk
T.M. Domżał, A. Stępień – *Zalety i ograniczenia w leczeniu migreny tryptanami / Advantages and restrictions on tryptans in migraine treatment*
A. Szczepańska-Szerej – *Hemikrania - problemy z rozpoznaniem / Hemicrania - diagnostic issues*
A. Prusiński – *Łagodna nawracająca „vestibulopatia" / Mild reccurent vestibulopathy*
J. Wojczal – *Diagnostyka ultrasonograficzna zawrotów głowy/ Ultrasonographic diagnostics of vertigo*

17.30 Otwarcie / Opening Ceremony
F. Gerstenbrand – *Apallic syndrome – new developments (Danube Lecture)*

19.15 Odjazd autobusów na kolację (szczegółowe informacje w dalszej części programu)
19.30 Fakultatywna kolacja w karczmie „Siwy Dym" w Celejowie