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]

[1] *Department of Neurology, Paracelsus Medical University Salzburg, Austria*
[2] *Neuroscience Institute, Paraceslus Medical University Salzburg, Austria*
[3] *Franz Tappeiner Hospital, Meran, Italy*
[4] *Department of Neurology, Medical University Innsbruck, Austria*
[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]

[1] *Department of Neurology, Paracelsus Medical University Salzburg, Austria*
[2] *Neuroscience Institute, Paraceslus Medical University Salzburg, Austria*
[3] *Center for Neurocognitive Research, Paris Lodron University Salzburg, Austria*
[4] *Franz Tappeiner Hospital, Meran, Italy*
[5] *Karl Landsteiner Inst. of Neurorehabilitation a. Space Neurol., Vienna, Austria*
[6] *Department of Neurology, Medical University Innsbruck, Austria*

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]

[1] *Department of Neurology, Medical University Innsbruck, Austria*
[2] *Karl Landsteiner Inst. of Neurorehabilitation a. Space Neurol., Vienna, Austria*
[3] *Department of Neurology, Paracelsus Medical University Salzburg, Austria*
[4] *Neuroscience Institute, Paracelsus Medical University Salzburg, Austria*
[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

POSTERS

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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Karl Landsteiner Institute of Neurorehabilitation and Space Neurology

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

SM. Golaszewski[1,4], M. Seidl [1,2], A.B. Kunz[1], M. Kronbichler[1], J. Bergmann [1], J. Crone [1], R. Nardone [3], E. Trinka[1], F. Gerstenbrand[4]
[1] Department of Neurology and Neuroscience Institute, Paracelsus Medical University, Salzburg
[2] TMS & fMRI Lab, Department of Neurology, Paracelsus Medical University, Salzburg, Austria
[3] Department of Neurology, Franz Tappeiner Hospital, Merano, Austria
[4] Karl Landsteiner Institute for Neurorehabilitation and Space Neurology, Vienna

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Dresden, Germany

Motivation for the study

Patients with severe chronic disorders of consciousness of different origin (TBI, hypoxia, stroke), Apallic Syndrome AS/VS (full state, early remission state I, II - Gerstenbrand 1967), patients in minimally conscious state are misdiagnosed up to 43% (Andrews et al, 1996; Schnakers et al, 2009)

Control procedure:

Bedside testing (neurological examination, Coma Recovery Scale - revised, CRS-R)

EEG (semantic oddball paradigm - SOP, own name paradigm - ONP)

fMRI (SOP, ONP)

Patient epidemiology and etiology of brain damage II

Patient	Etiology	Age	Gender	fMRI delay	CRS-R sum Σ
VS11	BS infarctions	39 years	male	1456 days	2
VS12	T hemorrhage	45 years	male	183 days	2
VS13	Hypoxia & astrocytoma II	38 years	male	66 days	6
VS14	T hemorrhage	38 years	male	344 days	4
VS15	Hypoxia	52 years	female	3 years	6
MCS1	T hemorrhage	77 years	male	33 days	9
MCS2	Hypoxia	19 years	male	95 days	9
MCS3	BS infarctions	59 years	male	86 days	15
MCS4	T hemorrhage	53 years	male	101 days	14
MCS5	T hemorrhage	46 years	male	5 years	8

T: traumatic, BS: brainstem

Coma Recovery Scale Revised (CRS-R) in bedside testing (BT)

Coma Recovery Scale Revised Score

#	auditory	visual	motor	oromotor	comm.	arousal	total
VS#1	1	0	0	1	0	1,5	3,5
VS#2	1	0	0	0	0	2	3
VS#3	1	1	1	0,5	0	1	4,5
VS#4	1,5	0	2	1	0	0	4,5
VS#5	1	0	0,5	1	0	0	2,5
VS#6	1	0	2	1	0	0	4
VS#7	2	1	2	1	0	1	7
VS#8	1	0	0	1	0	2	4
VS#9	1	0	0	1	0	1	3
VS#10	0	0	1	1	0	1	3
VS#11	0	0	1	1,5	0	0,5	3
VS#12	0,5	0	0,5	0	0	0	1
VS#13	1	1	1	1	0	2	6
VS#14	1	0	1	1	0	1	4
VS#15	1	0	2	1	0	2	6
MCS#1	1	3	1	1	0	3	9
MCS#2	1	2,5	1	1	1	2	8,5
MCS#3	4	3	3	1	1	3	15
MCS#4	2	3	4	2	1	2	14
MCS#5	1	2,5	2	1	0	1,5	8

Detailed anatomical analysis of the lesion pattern - 1

	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]		[illegible]				
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]		[illegible]		[illegible]		
[illegible]	[illegible]	laminar necrosis, moderate atrophy, biparietal gliosis	periventricular, to cortical U-Fibers	[illegible]	[illegible]					
[illegible]	multiple infarctions		[illegible]	slight dilatation of 3rd, 4th ventricle and perpontine and cerebellomedullar cisterns		cerebellar peduncli, left hemisphere	paramedian bilateral	complete bilateral infarction of pons including medial lemniscus, pyramidal tract, red nucleus, substantia nigra	degeneration of truncus	
UWS5	Hypoxia	laminar necrosis, slight atrophy	periventricular gliosis, microangiopathy	[illegible] ventricles, subarachnoidal; chronic transventricular CSF resorption	Caudate nuclei, putamen, microangiopathy			[illegible]		
UWS6	multiple infarctions		arteriosklerotic leukencephalopathy	slight dilatation of all ventricles, subarachnoidal and perpontine cisterns		small infarctions cerebellar peduncles, right cerebellar white matter			degeneration of truncus	
UWS7	Hypoxia	[illegible], biooccipital gliosis, moderate atrophy	periventricular, esp. bilat. posterior horns to cortical U-Fibers	slight dilatation of all ventricles, subarachnoidal and perpontine cisterns	caudate nuclei, putamen (bil)					
[illegible]	[illegible]		[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]		[illegible]	[illegible]	[illegible]	
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]					

Detailed anatomical analysis of the lesion pattern - 2

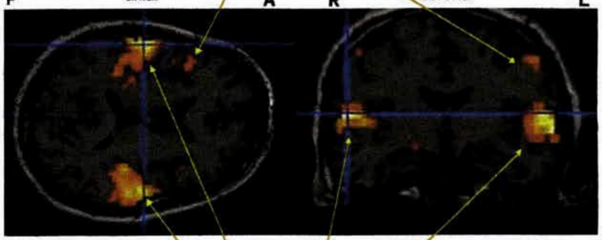
	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]		[illegible]		[illegible]	[illegible]	[illegible]		[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]				[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]				[illegible]	[illegible]	[illegible]
[illegible]	[illegible]		[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]			[illegible]	[illegible]	
[illegible]	[illegible]		[illegible]					[illegible]		
[illegible]	[illegible]	[illegible]	[illegible]			[illegible]		[illegible]		
[illegible]	[illegible]		[illegible]			[illegible]		[illegible]		
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]		[illegible]	[illegible]	
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	

Semantic Oddball paradigm (meaningful versus non-meaningful sentences)

e.g. The sun is hot

e.g. With the ears one can speak

SOP/fMRI: 44 y. old Patient, Locked-In-Syndrome plus severe hypersomnie post Basilar thrombosis 3 years ago



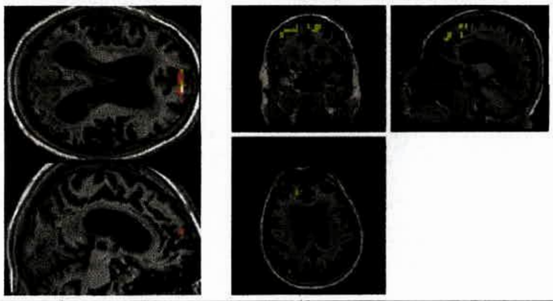
Own name paradigm (own versus other first name)

e.g. Markus, hello Markus ...

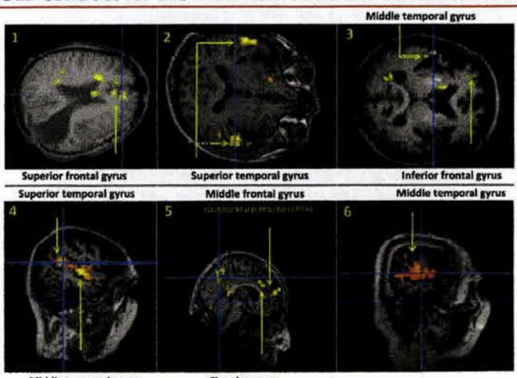
ONP/fMRI: patients

- Patient: 45 y. old
- Basilaris thrombosis 6 mo ago
- No response in bedside testing

- Patient: 50 y. old
- Hypoxic Encephalopathy post cardiac arrest 3 mo ago
- No response in bedside testing



BOLD contrast for the Own Name and the Sentence Paradigma



1) MCS 2: own name > not own name
2) UWS 11: own name > silence
3) UWS 3: sentences > silence
4) UWS 6: meaningful > non-meaningful
5) MCS 3: own name > silence
6) UWS 7: sentences > silence

Functional MRI paradigms: specific responses in 7 patients

Patient	Paradigm	GTs prim L	GTs prim R	GTs Wernicke's L	GTs Wernicke's R	GFi L	GFi R	GFm DLPFC L	GFm DLPFC R	GFs L	GFs R
UWS3	S>R	+				+					
	M>NM										
	O>R										
	O>NO										
UWS4	S>R	+	+		+						
	M>NM		+		+						
	O>R										
	O>NO						+	+	+	+	+
UWS6	S>R	+	+	+	+	+					
	M>NM		+		+		+				
	O>R	+	+	+	+						
	O>NO					+					
UWS11	S>R	+	+	+							
	M>NM										
	O>R	+	+								
	O>NO										
MCS1	S>R	+	+	+	+						+
	M>NM										
	O>R										
	O>NO	+	+	+	+						
MCS2	S>R										
	M>NM										
	O>R	+	+			+					
	O>NO					+	+				
MCS3	S>R	+									
	M>NM										
	O>R	+	+	+	+						
	O>NO										

Specific brain areas:

GTs prim: transvers temporal gyrus

GTs: Wernicke's superior temporal gyrus

GFi: Inferior frontal gyrus

GFm DLPFC: middle frontal gyrus, dorsolateral prefrontal cortex

GFs: superior frontal gyrus

GTm: medial temporal gyrus

Contrasts:

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

Funktionelle Neuroanatomie des SP und ONP - 1

fMRI passive listening	Paradigm	GTs prim L	GTs prim R	GTsWernicke's L	GTsWernicke's R	Gfi L	Gfi R	GFm DLPFC L	GFm DLPFC R	GFs L	GFs R	Other
UWS2	S>R											
	M>NM					+						
	O>R											
	O>NO											
UWS3	S>R	+				+						
	M>NM											
	O>R											
	O>NO											
UWS4	S>R	+	+		+							
	M>NM		+		+							
	O>R	+	+	+	+							
	O>NO						+	+	+	+	+	left precuneus, left BA 17, left insula
UWS5	S>R	+	+	+	+							
	M>NM											
	O>R	+	+	+	+							
	O>NO											
UWS6	S>R	+	+	+	+	+						right precentral gyrus
	M>NM		+		+		+					
	O>R	+	+	+	+							precuneus, cingular gyrus, BA 17
	O>NO					+						superior parietal lobule, precuneus
UWS7	S>R											
	M>NM											
	O>R	+	+					+	+		+	precuneus, cingular gyrus
	O>NO											
UWS8	S>R		+									
	M>NM											right inferior temporal gyrus
	O>R		+									
	O>NO									+		

Funktionelle Neuroanatomie des SP und ONP - 2

fMRI passive listening	Paradigm	GTs prim L	GTs prim R	GTsWernicke's L	GTsWernicke's R	Gfi L	Gfi R	GFm DLPFC L	GFm DLPFC R	GFs L	GFs R	Other
UWS11	S>R	+	+	+								
	M>NM											
	O>R	+	+									
	O>NO											
UWS13	S>R	+										
	M>NM											
	O>R											
	O>NO											
UWS14	S>R	+	+	+	+							
	M>NM											
	O>R	+	+	+	+							BA 17, fusiforme gyrus
	O>NO									+		
MCS1	S>R	+	+	+	+							
	M>NM											
	O>R											left GTm
	O>NO	+	+	+	+							
MCS2	S>R											
	M>NM											
	O>R	+	+			+						
	O>NO					+	+					bilateral medial prefrontal cortex
MCS3	S>R	+										
	M>NM											
	O>R	+	+	+	+							
	O>NO											
MCS4	S>R	+	+	+	+							
	M>NM											
	O>R	+	+	+	+							
	O>NO											
MCS5	S>R	+	+	+	+							
	M>NM											
	O>R	+		+								
	O>NO											

Results I: fMRI/EEG, AS patients in bedside testing

patient number	vibrotactile stimulation	silence vs name	own name vs foreign name	silence vs sentence	semantic oddball
VS#1	no	no	no	no	no
VS#2	no	no	yes	yes	no
VS#3	no	no	no	yes	no
VS#4	yes	yes	yes	yes	yes
VS#5	no	yes	no	yes	no
VS#6	yes	yes	yes	yes	yes
VS#7	no	yes	no	no	no
VS#8	no	yes	yes	yes	yes
VS#9	yes	no	no	no	no
VS#10	yes	no	no	no	no
VS#11	no	yes	no	yes	no
VS#12	yes	no	no	no	no
VS#13	yes	no	no	yes	no
VS#14	no	yes	yes	yes	no
VS#15	no	no	no	no	no

Results II: fMRI/EEG, MCS patients in bedside testing

patient number	vibrotactile stimulation	silence vs name	own name vs foreign name	silence vs sentence	semantic oddball
MCS#1	no	yes	yes	yes	no
MCS#2	no	yes	yes	yes	yes
MCS#3	no	yes	no	yes	no
MCS#4	on	yes	no	yes	yes
MCS#5	no	yes	yes	yes	no

⇒ 8 out of the 15 AS patients in BT diagnosis did show higher order speech processing and cortical response to a self-referential stimulus in fMRI

Discussion

The best possible diagnoses and prognoses as accurate as possible are essential for the justification of medical, legal and ethical reasons for rehabilitation measures as follows:

- Improvement of the rehabilitation result (identification of programs for a possible rehabilitation)
- To give the patient the opportunity to express their condition (e.g. pain, state of mind)
- Give patients the opportunity to express their will (e.g. last will, end of life decisions, etc.)

Conclusion

In unresponsive patients diagnosed as Apallic Syndrome (AS/VS) BT fMRI shows specific brain activity in language regions and regions of self-awareness. EEG shows a differential response to sentences and names. It can be concluded that the diagnosis of AS in BT has to be revised, patients are able for the processing of language, memory and self-referential stimuli at a higher cortical level.

fMRI and EEG showed consistent results.

Knowledge about the perception of language and self-referential stimuli in patients with severe disorders of consciousness is very important for individual planning of neurorehabilitation program and for relatives, caregivers and therapists to improve outcome.

Up to now, we do not have any data for the prognostic value of the detected specific brain activity in fMRI and EEG. Thus, long-term assessment s for AS and MCS patients in BT are needed.


Thank you for your attention!