THE SOCIETY FOR THE STUDY OF NEUROPROTECTION AND NEUROPLASTICITY

Academia de Ştiinţe Medicale din România

UMF UNIVERSITATEA DE MEDICINĂ ŞI FARMACIE IULIU HAŢIEGANU CLUJ-NAPOCA

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snr societatea de neurologie din romania

7TH CONGRESS OF THE SOCIETY FOR THE STUDY OF NEUROPROTECTION AND NEUROPLASTICITY

MARCH 31 / APRIL 3 / 2011

KRAKOW | POLAND

FINAL PROGRAM AND ABSTRACT BOOK

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CONGRESS VENUE

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GENERAL INFORMATION

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CONGRESS REGISTRATION DESK
All materials and documentation will be available at the registration desk located at SSNN booth. The staff will be pleased to help you with all enquiries regarding registration, materials and program. Please do not hesitate to contact the staff members if there is anything they can do to make your stay more enjoyable.

OPENING HOURS
Thursday 31 March 2011 | 19:00 - 20:30
Friday 1 April 2011 | 8:30 - 19:30
Saturday 2 April 2011 | 8:30 - 19:30
Sunday 3 April 2011 | 8:30 - 12:00

REGISTRATION FEE:

September 1st 2010 - December 1st 2010 : 300 EUR
December 2nd 2010 - March 30th 2011: 400 EUR
On-site registration: 450 EUR

PARTICIPANTS REGISTRATION FEE INCLUDES:

Admission to all scientific sessions during the congress.
Conference materials (delegate bag, final program and abstract book etc.)
Admission to Lunches and Coffee Breaks.

ON-SITE REGISTRATION

On-site registration will be processed on a first-come, first-served basis. Priority will be given to pre-registered delegates. Depending on the number of on-site registered delegates, availability of congress bags may be limited.

CONGRESS LANGUAGE

The congress language is English. Simultaneous translation will not be provided.

CHANGES IN PROGRAM

The organizers cannot assume liability for any changes in the congress program due to external or unforeseen circumstances.

ABSTRACT BOOK

The participants documents include the abstract book which will be handed out together with the congress bag at the registration counter.

CONTACT

If you need further information regarding technical details, please contact:
Ovidiu Selejan/e-mail/ovidius@ssnn.ro For updates and details please visit our website/www.ssnn.ro

COFFEE BREAKS, LUNCHES, DINNERS

THURSDAY / 31st March 2011

19:30 - Welcome Reception | Olive Restaurant (Sheraton Hotel)

FRIDAY / 1st April 2011

10:00 – 10:20 Coffee Break
13:40 – 14:30 Lunch | Olive Restaurant (Sheraton Hotel)
16:00 – 16:30 – Coffee break

20:45 – Gala Dinner | Olive Restaurant & Wisla Ballroom (Sheraton Hotel)

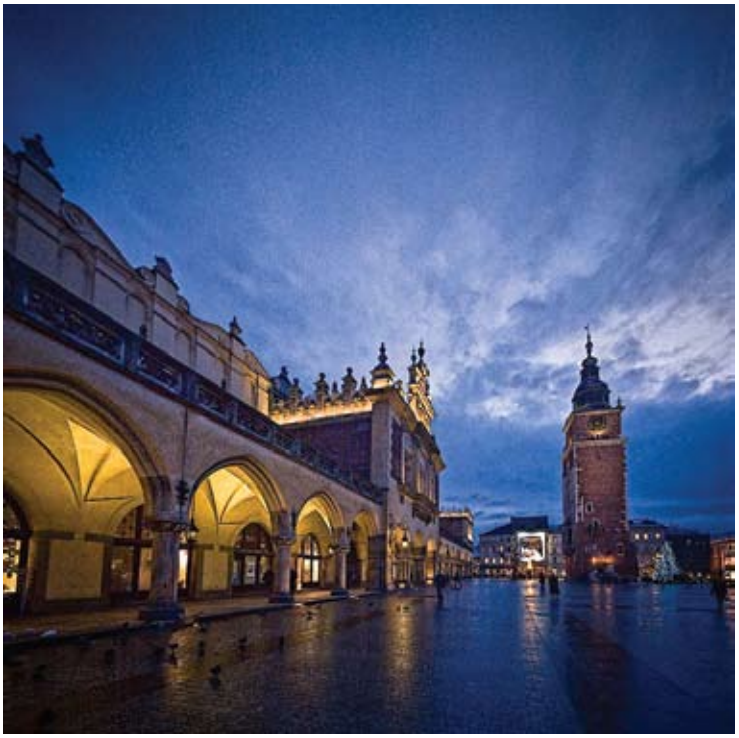
SATURDAY / 2nd April 2011

11:10 – 11:30 Coffee break
14:00 – 15:00 – Lunch | Olive Restaurant (Sheraton Hotel)
16:40 – 17:00 – Coffee Break

20:30 – Gala Dinner
| Olive Restaurant & Wisla Ballroom (Sheraton Hotel)

SUNDAY / 3rd April 2011

10:30 – 11:00 Coffee Break
13:00 Lunch | "Some Place Else" Restaurant (Sheraton Hotel)



SCIENTIFIC PROGRAM

Scientific Program
7th Congress of the Society for the Study of Neuroprotection and Neuroplasticity
Krakow, March 31st – April 3rd, 2011

FRIDAY / 1st April 2011

08:15 – 08:30 – Welcome Address (Dafin F. Mureşanu, Natan Bornstein, Vladimir Hachinski, Hari Shanker Sharma, Anna Członkowska)

Presidential Session

08:30 – 09:00
Dafin F. Mureşanu (Romania) | Pathological neuroplasticity in impulse control disorders

09:00 – 09:30
Natan Bornstein (Israel) | Stress and stroke

09:30 – 10:00 - Special lecture by Vladimir Hachinski (Canada) | Subclinical strokes and insidious Alzheimer disease: a new frontier in prevention?

10:00 – 10:20 Coffee Break

Session 1 – Stroke
Chairperson: Anna Członkowska, Paule Merle

10:20 – 10:40
László Csiba (Hungary) | Interventions in acute ischemic stroke

10:40 – 11:00
Vida Demarin (Croatia) | Stroke and neuroplasticity

11:00 – 11:20
Roberto Maturana (Chile) | Neuroplasticity and brain repair after stroke

11:20 – 11:40
Exuperio Díez-Tejedor (Spain) | Brain repair therapies in ischemic stroke. The role of trophic factors and stem cells

11:40 – 12:00
László Vécsei (Hungary) | Novel therapeutic strategies of stroke and neurodegenerative disorders: the role of kynurenines

Discussions – 12:00 – 12:10

12:10 – 13:40

Chairmen - Natan Bornstein, Dafin F. Mureşanu

Michael Chopp (USA) | Neurotrophic factors and remodeling of ischemic brain
Natan Bornstein (Israel) | Lesson learned from CASTA trial and future perspectives
Volker Hömberg (Germany) | Pharmacological support of neurorehabilitation

Dafin F. Mureşanu (Romania) - Conclusions and comments

13:40 – 14:30 Lunch

Session 2 – Traumatic Brain Injury
Chairperson: Antonio Federico, Harry Steinbusch

14:30 – 14:50
Pieter E. Vos (The Netherlands) | Moderate and severe traumatic brain injury: a prospective multicentre cohort study

14:50 – 15:10
Bogdan O. Popescu (Romania) | Early neurotrophic factor treatment in traumatic brain injury – a large retrospective, national, multicenter cohort study

15:10 -15:30
Beata Sániová (Slovakia) | Neuroprotective therapeutic results in comatous patients with amantadin sulfate

15:30 – 15:50
Klaus von Wild (Germany) | Unresponsive wakefulness syndrome (UWS) - Proposal for a new terminology of apallic syndrome / vegetative state

15:50 – 16:00 - Discussions

16:00 – 16:30 – Coffee break

16:30 – 18:30 Special Parallel Session: Ibero-American Stroke Experts Workshop
Chairman - Exuperio Díez-Tejedor (Spain)
Venue: Warszawa Meeting Room

Session 3 – Neurorehabilitation (I)
Chairperson: Volker Hömberg, Pieter E. Vos

16:30 – 16:50
Heinrich Binder (Austria) | Critical illness: early rehabilitation of sequelae essential

16:50 – 17:10
Anna Członkowska (Poland) | Dose BDNF -196 G>A and -270 C>T polymorphisms is associated with clinical course of multiple sclerosis and stroke recovery?

17:10 – 17:30
Gelu Onose (Romania) | Our experience with neurorehabilitation TBI patients, treated with neurotrophic factors

Discussions – 17:30 – 17:40

Session 4 – Neurorehabilitation (II)
Chairperson: Heinrich Binder, Klaus von Wild

17:40 – 18:00
Volker Hömberg (Germany) | Future aspects of motor therapies in neurorehabilitation

18:00 – 18:20
C.D. Popescu (Romania) | Electrophysiological disfunction of corpus callosum in patients with multiple sclerosis

18:20 – 18:40
Maciej Krawczyk (Poland) | Recent advances in neurorehabilitation

18:40 – 18:50 – Discussions

20:45 – Welcome Reception

SATURDAY / 2nd April 2011

Session 5 – Dementia and Neurodegenerative Disorders (I)
Chairperson: Rudolph Castellani, Laszlo Csiba

08:30 – 08:50
Amos Korczyn (Israel) | Why have we failed to cure Alzheimer's disease?

08:50 – 09:10
Anton Alvarez (Spain) | Trophic factors: role in AD pathology and therapy

09:10 – 09:30
Ovidiu Băjenaru (Romania) | What are the targets for potential neuroprotection in multiple sclerosis?

09:30 – 09:50
Stavros J. Baloyannis (Greece) | The Golgi apparatus in Alzheimer's disease

09:50 – 10:00 Discussions

Session 6 – Dementia and Neurodegenerative Disorders (II)
Chairperson: Amos Korczyn, Stephen Skaper

10:00 – 10:20
Raul Arizaga (Argentina) | Neurodegeneration vs neuroprotection - the cognitive and behavioral impairment equation

10:20 – 10:40
Peter Riederer (Germany) | Risk factors for cognitive decline and dementia

10:40 – 11:00
Emil C. Toescu (UK) | Increased neuronal vulnerability in normal brain ageing

11:00 – 11:10 Discussions

11:10 – 11:30 Coffee break

11:30 – 12:30

Amos Korczyn (Israel) | Do we need to redefine PD?

Ovidiu Băjenaru (Romania) | Utility of the PDSS score for evaluating patients with Parkinson's disease

Dafin F. Mureşanu (Romania) | New approaches in long term care management of Parkinson's disease

Session 7 – Mood and Cognitive Disorders
Chairperson: Russell Andrews, Mohamed El Tamawy

12:30 – 12:50
Johannes Thome (UK) | Neurotrophic factors and synaptic vesicle proteins: a common pathophysiology in depression and dementia?

12:50 – 13:10
Boris Kotchoubey (Germany) | Attentional mechanisms of mood control: Neuro-physiological correlates of mindfulness in recurrent depression

13:10 – 13:30
Jeffrey Schwartz (USA) | Mindful awareness, quantum mechanics and self-directed neuroplasticity

13:30 – 13:50
Simone Lang (Germany) | Neural correlates of empathy to pain in patients with disorders of consciousness

13:50 – 14:00 Discussions

14:00 – 15:00 – Lunch

Session 8 – Basic Research (I)
Chairperson: Johannes Thome, Bogdan O. Popescu

15:00 – 15:20
Harry Steinbusch (The Netherlands) | Prevention of age-related changes in hippocampal levels of 5- methylcytidine by caloric restriction

15:20 – 15:40
Hari Shanker Sharma (Sweden) | Superior neuroprotective effects of neurotrophic factors in heat stroke following nanoparticles treatment

15:40 – 15:50 Discussions

Session 9 – Basic Research (II)
Chairperson: Peter Riederer, Anton Alvarez

15:50 – 16:10
Paule Merle (USA) | Neonatal ketamine anesthesia, neuronal cell death and long-lasting cognitive deficits in nonhuman primates

16:10 – 16:30
Ştefan Florian (Romania) | Complex investigation of angiogenesis in Glioblastoma Multiforme stem cells

Discussions – 16:30 – 16:40

Coffee Break – 16:40 – 17:00

Session 10 – Neurotrophic factors - new therapeutic approaches
Chairperson: Ovidiu Băjenaru, Ştefan Florian

17:00 – 17:20
Antonio Federico (Italy) | Mitochondrial functions and dysfunctions in some inherited neurological diseases as a model of neuroregeneration and neuroprotection

17:20 – 17:40
Tomasz Gaszynski (Poland) | Neurotrophic factors in treatment of consciousness disturbances in ICU and its influence on brain tissue oxygenation measured with NIRS monitor

17:40 – 18:00
Cornel Cătoi (Romania) | Neuromodulation following connection of the first motor neuron to the peripheral nerve stump via grafting in rats

18:00 – 18:20
Andrzej Glabinski (Poland) | Neuroinflammation and neurodegeneration in mul-tiple sclerosis and its experimental models

Discussions 18:20 – 18:30

20:30 – Gala Dinner

SUNDAY / 3rd April 2011

Session 11 – Basic Research (III)
Chairperson: Hari Shanker Sharma, Raul Arizaga

08:30 – 08:50
Stephen Skaper (Italy) | Amyloid and Alzheimer's Disease

08:50 – 09:10
Russell J. Andrews (USA) | Future neuroprotection and neuroplasticity: from 'shotgun' drugs and electrodes to 'laser-precise' electrochemical therapeutics

09:10 – 09:30
Rudolph Castellani (USA) | Granulovacuolar Degeneration is the human homologue to stress granules: implications for Alzheimer 's disease pathogenesis

09:30 – 09:40 – Discussions

Session 12 – Epilepsy
Chairperson: Anwar Etribi, Emil Toescu

09:40 – 10:00
Anca Buzoianu (Romania) | Genotype-phenotype correlations between the alleles of the CYP2C19 polymorphism and pharmacokinetic parameters in Romanian epileptic patients

10:00 – 10:20
Hassan Hosny (Egypt) | Subtypes of nonconvulsive status epilepticus

10:20 – 10:30 – Discussions

Coffee Break – 10:30 – 11:00

Session 13 – New approaches in neurorehabilitation
Chairperson: Hassan Hosny, A. V. Ciurea

11:00 – 11:20
Mohamed El-Tamawy (Egypt) |Brain death: Towards an active diagnosis

11:20 – 11:40
A.V. Ciurea (Romania) | Timing of neuroprotection and neurorecovery in acute traumatic brain injuries (TBI)

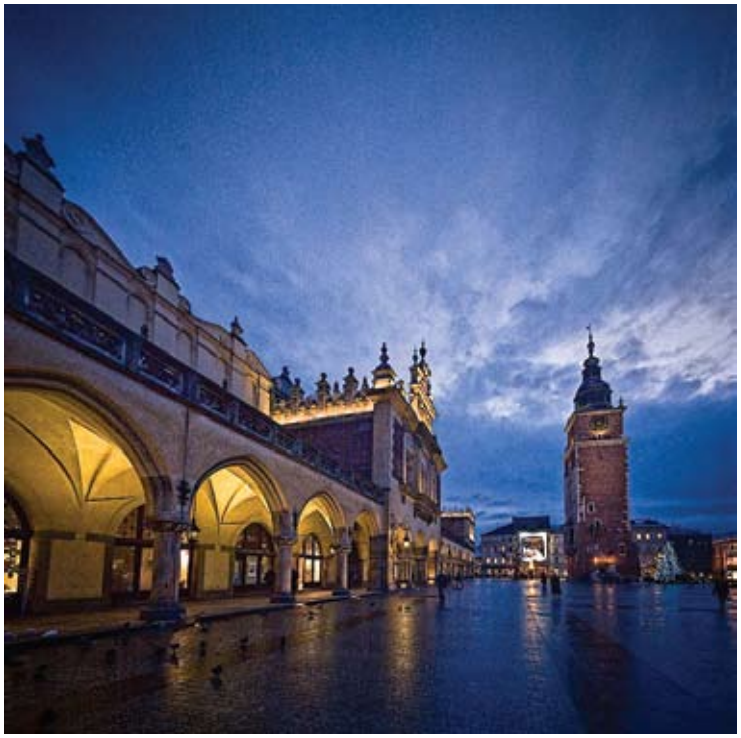
11:40 – 12:00
Awar Etribi | The effect of neuroprotection and repetitive transmagnetic stimulation in stroke patients.

12:00 – 12:20
Józef Opara (Poland) | Novel methods of post-stroke rehabilitation enhancing use-dependent plasticity of brain

12:20 – 12:30 – Discussions

12:30 – 13:00 – Closing remarks – Dafin F. Mureşanu, Natan Bornstein
Hari Shanker Sharma

13:00 Lunch



ABSTRACTS

TROPHIC FACTORS: ROLE IN AD PATHOLOGY AND THERAPY

ANTON ALVAREZ

Department of Neuropharmacology, EuroEspes Biomedical Research Centre, A Coruña, Spain

A deficit of brain trophic activity seems to constitute an early event in the ethipathogenesis of Alzheimer's disease (AD). Downregulations of nerve growth factor (NGF), brain derived neurotrophic factor (BDNF) and insulin-like growth factor-I (IGF-I) have been reported in AD and in mild cognitive impairment (MCI). The expression of the high affinity NGF receptors (TrkA) is reduced in cholinergic neurons of the nucleus basalis of Meynert and in cortical regions of both AD and MCI brains. Cortical BDNF and proBDNF levels and the expression of BDNF receptors (TrkB) into the nucleus basalis of Meynert are also reduced in AD and MCI. Decreased IGF-I expression and receptor binding have also been reported in AD brains. All these findings are relevant because: (1) NGF is essential for the survival of cholinergic neurons into the basal forebrain; (2) BDNF is responsible for neuronal plasticity and synaptogenesis, particularly in the hippocampus; and (3) IGF-I exerts pleiotropic effects into the brain and its blockade induces amyloid- and tau-related AD pathology.

Several experimental studies are in support of the therapeutic use of trophic factors in AD: (1) NGF and BDNF, through Trk signaling, prevent the amyloidogenic cleavage of APP and the apoptotic death of neurons; (2) transgenic BDNF expression prevents neuro-degeneration in various animal models of AD; (3) IGF-I reduces brain amyloid pathology in transgenic AD mice models. Some treatment approaches with neurotrophic factors showed also clinical efficacy in AD patients.

Cerebrolysin is a brain-derived neurotrophic preparation with pleiotropic activity. In experimental conditions it displays NGF- and BDNF-like neurotrophic ef-fects, protecting against several components of the AD pathological cascade: has anti-excitotoxic, antiapoptotic and antiinflammation effects; reduces APP phosphorylation, cerebrovascular amyloidosis and tau pathology by modulating GSK3β and CDK5 activity; and promotes neuronal survival, synaptogenesis and neurogenesis. Clinical studies demonstrated the safety and the efficacy neurotrophic factors monotherapy in AD, and support its suitability for combined AD therapy. Results of a recent trial indicate that the combined therapy with trophic and cholinergic drugs might provide long-term clinical benefits for AD patients. The synergistic effect of this combination treatment on AD clinical outcome is consistent with its influence on the circulating levels of trophic factors (BDNF and VEGF, vascular endothelial growth factor). These findings provide new insights on the relevance of neurotrophic factors in AD therapy.

FUTURE NEUROPROTECTION AND NEUROPLASTICITY: FROM 'SHOTGUN' DRUGS AND ELECTRODES TO 'LASER-PRECISE' ELECTROCHEMICAL THERAPEUTICS

RUSSEL J. ANDREWS

Ames Associate
Smart Systems & Nanotechnology
NASA Ames Research Center
Moffett Field, CA, USA

Techniques for modifying or repairing brain function have traditionally been either chemical or electrical. One might give a medication (usually orally or intravenously) - which, to the degree the drug crossed the blood-brain barrier, would typically result in diffuse brain tissue levels of the drug. Alternatively, one might administer an electrical stimulus - globally in the case of electroconvulsive therapy (ECT) and somewhat more focally in the case of deep brain stimulation (DBS) or spinal cord stimulation (SCS). However, the brain communicates by an electrochemical marriage, with the exchanges occurring at the cellular level - different neurotransmitters signaling side-by-side, and axons from different neurons intermingling at the micron level.

Devices that can stimulate and record both electrical and chemical activity in the brain with cellular level precision are in development. Microelectrodes and nanoarrays can monitor both electrical activity and neurotransmitter levels simultaneously and in virtually real-time. By focally modulating either electrical activity or neurotransmitter levels (or both), and monitoring either electrical activity or neurotransmitter levels (or both) – both in proximity to the stimulus and in remote regions of the brain - we can observe the response of the brain to 'real-world' precise interventions. This is quite an advance from the 'sledgehammer' techniques of diffuse drugs or macroelectrodes.
Some of these precision multifunctional techniques will be described, and the possibilities for enhancing neuroprotection and neuroplasticity using these techniques will be considered.

NEURODEGENERATION VS NEUROPROTECTION - THE COGNITIVE AND BEHAVIORAL IMPAIRMENT EQUATION

RAUL ARIZAGA

Cognitive Neurology Unit, Neuraxis Institute, Neurological Foundation, Buenos Aires, Argentina

Multiple factors influence the development of cognitive and / or behavioral impairment in an individual. Numerous epidemiological studies have identified risk and protective factors. At the same time, basic research has elucidated the different mechanisms involved both in intrinsic neuronal damage and in the transduction of the protective factors evidenced by epidemiology. The aim of this presentation is to summarize the interaction of these mechanisms. Only an approach that covers all aspects of the multivariate balance between neurodegeneration and neuroprotection will be useful when thinking of prediction, prevention and treatment tools for this group of chronic age related diseases.

THE GOLGI APPARATUS IN ALZHEIMER'S DISEASE

STAVROS J. BALOYANNIS

1st Department of Neurology, Aristotelian University, Thessaloniki, Greece

Alzheimer's disease (AD) is a devastating disorder of the central nervous system, involving progressive cognitive impairment, characterized mostly by marked memory loss, learning inability,loss of professional skills, behavioral changes, alterations of the personality resulting eventually in a vegetative state. The etiopathological background of AD, in spite of the continuous ongoing research efforts, remains still unknown. A cascade of pathogenetic factors may contribute in some extend, each one of them in plotting the neuropathological and clinical profile of the disease. The implication of ß-amyloid, APP, ßau protein, the neuronal loss, the synaptic changes, the mitochondrial alterations and the cytoskeletal alterations, play a substantial role in the formation of the morphological hallmarks of AD, without explaining sufficiently the innermost pathogenetic mechanisms of the disease. Morphological alterations of the neuronal organelles, mostly concerning the mitochondria, have been described in AD. histochemically as well as ultrastructurally. In the present study we attempted to describe the morphological alterations of the Golgi apparatus (GA) in twelve cases of AD, post mortem material, promptly fixed and studied in electron microscopy. It is known that GA, plays an important role in glycosilation, sulfation, trafficking and proteolytic processing of protein systems, synthesized in the endoplasmic reticulum of neurons and glial cells. The hyperphosphorylation of ßau protein, a phenomenon tightly associated with the pathogenesis of AD, is also related with the pathophysiology of GA. In our studied cases of Alzheimer's disease we observed a marked fragmentation of GA in the perikaryon of polyhedral and pyramidal cells of the hippocampus, the large triangular neurons of the frontal cortex as well as the Purkinje cells of the cerebellum. The fragmentation of Golgi apparatus, which was mostly associated with mitochondrial alterations and the loss of dendritic spines in most of the large polyhedral and pyramidal neurons, may be related with the action of ß secretase on APP, or it may be associated with the alterations of microtubules, since those cytoskeletal elements play an important role in spacial organization as well as the integrity of the cisternae of the Golgi apparatus.

Busciglio J, Gabuzda DH, Matsudaira P, Yankner BA Generation of beta-amyloid in the secretory pathway in neuronal and nonneuronal cells Proc Natl Acad Sci U S A 1993 Mar 1;90(5):2092-6

Salehi A, Heyn S, Gonatas NK, Swaab DF Decreased protein synthetic activity of the hypothalamic tuberomamillary nucleus in Alzheimer's disease as suggested by smaller Golgi apparatus.Neurosci Lett 1995;193:29-32

Salehi A, Lucassen PJ, Pool CW, Gonatas NK, Ravid R, Swaab DF Decreased neuronal activity in the nucleus basalis of Meynert in Alzheimer's disease as suggested by the size of the Golgi apparatus.Neuroscience 1994;59:871-80

Stieber A, Mourelatos Z, Gonatas NK. In Alzheimer's disease the Golgi apparatus of a population of neurons without neurofibrillary tangles is fragmented and atrophic. Amer J Path 1996;148:415-26Terry RD. The pathogenesis of Alzheimer disease: An alternative to the amyloid hypothesis. J Neuropathol Exp Neurol 1996;55:1023-25

WHAT ARE THE TARGETS FOR POTENTIAL NEUROPROTECTION IN MULTIPLE SCLEROSIS

OVIDIU BĂJENARU

University Hospital of Emergency Bucharest, Department of Neurology, „Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Multiple sclerosis is a complex chronic inflammatory and neurodegenerative disease of CNS, quite heterogenous both clinically and pathogenetic. The recent data on pathogenetic pathways of this disease suggest that early focal inflammatory changes in the blood-brain barrier and the complex cascade of both inflammatory-immune and neurodegenerative events develop together in a complex interrelated pathogenetic process, generating lesions leading to brain and spinal cord atrophy, clinically expressed as physical and mental impairment – often invalidating and very severe. The irreversible lesions in MS are the neurodegenerative changes implying not only axons and neurons but also the glial cells, while the inflammatory - demyelinating lesions, at least during the early phases of the disease are potentially reversible and they may trigger spontaneous neuroprotective mechanisms. Understanding the details of these complex pathogenetic pathways, it is reasonable that treatments adressing to early inflammatory events both at the blood-brain barrier and in the CNS tissue, facilitating early remyelination and not impairing the protective inflammatory pathway initiated mainly by Th2 cells, could have also indirect neuroprotective potential effects, also suggested by the clinical results of more recent trials (slowing the brain atrophy process, improvement of functional clinical scores, etc.). This approach is extremely important for everyday clinical practice today as long as we still not have a proven therapeutic possibility to stop in a direct manner the neurodegenerative changes present in this disease.

CRITICAL ILLNESS: EARLY REHABILITATION OF SEQUELAE ESSENTIAL

HEINRICH BINDER

Landsteiner Institute for Neurorehabilitation and space medicine
Vienna, Austria

The term critical illnes, although poorly defined, is widespread used. It contains a wealth of neuropsychiatric signs and symptoms mostly occuring during and after intensive care in relation to various serious diseases of various organs including the nervous system. Critical illness is a challenge for prevention, treatment and especially Rehabilitation - for prevention and therapy, as there are a plethora of metabolic and immunological factors that must be observed and for rehabilitation, because of long lasting disabling consequences.

STRESS AND STROKE

NATAN BORNSTEIN

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Head of Stroke Unit at the
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Chairman of the Israeli Neurological Association

Stroke is a stressful, life-threatening experience. The stress is intensified by the fact that stroke is often a sudden and unanticipated event over which the victim has no control.

In addition to the damage to the brain, the patient's sense of wholeness and safety might be shattered, leaving him or her with a lasting sense of vulnerability.

While the psychological impact of stroke is receiving increasing clinical attention, the underlying pathophysiology is poorly understood. Most likely stress in the acute and post-acute phase of stroke plays an important role, for example, by mediating post-traumatic stress disorder (PTSD)-like symptoms.

Recent evidence points to stress as a major determinant of subsequent clinical deterioration.
The psychological symptoms may be a direct consequence of focal lesions, but they may also be an indirect consequence of psychological stress which frequently occurs during an acute stroke.

At the psychological level, any life threatening experience accompanied by an emotional response including intense fear, helplessness or horror may lead to various PTSD-like symptoms. The cognitive deterioration associated with PTSD in numerous neuropsychological studies include impaired learning and recall, verbal memory, working memory, attention, and self-referential semantic processing.

The hypothalamic–pituitary–adrenal (HPA) axis is a significant portion of the neuroendocrine system and is largely responsible for controlling stress reactions and affect regulation. Cortisol, the major stress hormone, is an output of the HPA axis with binding affinity for cerebral glucocorticoid (GC) receptors. Limbic regions, such as the hippocampus, amygdala and prefrontal cortex (PFC) are targeted by glucocorticoids and are essential to produce adapted and
integrated neuroendocrine and behavioral responses to stress. "Targets" of stress on a neural systems level include the hippocampus, the amygdala, and the PFC.

Acute stress (via increased excitatory amino acids, reduced BDNF, and/or increased GCs), is associated with inhibition of hippocampal neurogenesis and loss of neuronal branching in the hippocampus. These effects are reversed by a variety of antidepressant treatments.
Acute stress elicits a transient increase in the amounts released of the neurotransmitter acetylcholine and a phase of enhanced neuronal excitability.

Finally, the acute phase of stroke is associated with increases in circulating pro-inflammatory cytokines and proinflammatory pathways, with recent studies demonstrating

causal links between inflammatory pathways and cholinergic signaling. The cholinergic anti-inflammatory pathway may be mitigated by acetylcholinesteras (AChE) and the closely related enzyme butyrylcholinesterase (BChE), both of which hydrolyze and inactivate acetylcholine.
We have previously shown that serum AChE and Cholinergic Status measurements can predict neurological outcome, survival and inflammatory reactions following acute ischemic stroke.

Recently, in a preliminary analysis, we found inverse correlation between hippocampal volume and bedtime saliva cortisol levels. Stroke patients in the upper tertile of bedtime cortisol had smaller hippocampal volume than those in the lower tertile and presented inferior cognitive scores.

The stress-related (PTDS-like) mechanisms could have particularly profound long-term consequences when they meet an already damaged brain after an ischemic stroke.
Therefore, stress, as well as secondary hippocampal/limbic lesions induced by the stressful life-event, might be devastating contributors to a poor post-stroke prognosis.

The “stressogenic vulnerable patient” is prone to develop anxiety symptoms and cognitive impairment.
Hence, early detection of maladaptive stress response in stroke patients has the potential to provide new concepts for targeted intervention aimed at slowing the pace of post-stroke deterioration of cognitive and affective function.

GENOTYPE-PHENOTYPE CORRELATIONS BETWEEN THE ALLELES OF THE CYP2C19 POLYMORPHISM AND PHARMACOKINETIC PARAMETERS IN ROMANIAN EPILEPTIC PATIENTS

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Abstract: The aim of the study was to evaluate the influence of genetic status on the metabolism of valproic acid (VPA) and to make the correlation between the genotype and the phenotype of the metabolizing status of the patients, measured in plasma levels of VPA.

Materials and method: 80 patients with a mean age of 39.25±1.59, either with idiopathic or secondary epilepsy, evaluated in the Neurology Clinic of Cluj-Napoca were included. Steady state plasma concentration of VPA were determined using the GC/FID technique to all patients under a stable treatment for at least 1 month. We considered therapeutic level between 50-100 ←g/mL. Using the PCR-RFLP method we've determined allelic variant of CYP2C19*2 and CYP2C19*3.

Results: 62% of the patients had therapeutic level of VPA, while 20% had sub-therapeutic and 18% of the patients supra-therapeutic level of it. 22.5% of the patients were heterozygous for CYP2C9*2, and 1.25% were homozygous, while 21.25% of the patients were heterozygous for CYP2C9*3. Regarding CYP2C19*2 16.25% of the patients were heterozygous and 7.5% of them homozygous. The polymorphism of CYP2C19*3 was absent. There were no significant correlation between the genetic status and the plasma concentrations of VPA.

Conclusions: The different allelic expression of CYP2C9 and CYP2C19 have no statistically significant influence on plasmatic level of VPA.

Key words: pharmacogenetics, CYP, valproic acid

GRANULOVACUOLAR DEGENERATION IS THE HUMAN HOMOLOGUE TO STRESS GRANULES: IMPLICATIONS FOR ALZHEIMER'S DISEASE PATHOGENESIS.

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The phosphorylated ribosomal protein S6 (pS6) is associated with the 40S ribosomal subunit in eukaryotes and is thought to play a role in RNA storage, degradation, and re-entry into translation. In our recent study, we found pS6 localized to granulovacuolar degeneration within pyramidal neurons. Immunohistochemical analysis found nearly 20 fold more neurons contain pS6 positive granules in Alzheimer's disease (AD) hippocampus compared with age-matched controls. Further, pS6-positive granules were found more often in neurons not containing neurofibrillary tangles, were never associated with extracellular neurofibrillary tangles, and contained less RNA than neighboring pyramidal neurons not containing pS6. In other model systems, this protein is a specific marker for stress granules, which are transient intracellular dense aggregations of proteins and RNAs that accumulate as a stress response, protecting cells from apoptosis and inappropriate transcriptional activity, as a form of "molecular triage." Since chronic oxidative stress is central to AD pathogenesis, and RNA is a specific oxidative stress target and is intimately associated with stress granule biogenesis in model systems, we suggest that granulovacuolar degeneration in human brain parallel stress granules, and may in fact be more representative of disease pathogenesis than traditionally believed. This proposed novel origin for GVD may represent a morphologic checkpoint between cell death and reversible cellular stress that proceeds in the absence of other inclusions.

NEUROMODULATION FOLLOWING CONNECTION OF THE FIRST MOTOR NEURON TO THE PERIPHERAL NERVE STUMP VIA GRAFTING IN RATS.

CORNEL CĂTOI

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Objective: Pharmacological neuroprotective and neuromodulating effects become crucial for restorative surgery to regain motor functioning in human spinal cord injuries and brachial plexus avulsion. New operative techniques and pharmacological treatment modalities need well designed and carefully observed experimental studies in animals. Close co-operation of veterinarian and human medicine scientists and personal is crucial for planning and realization of the protocols in animal experiments according to international law and standards for best practice. This is the case in our study on the functional re-innervation and neuroprotection through peripheral nerve graft connecting of the spinal cord to a rat skeletal muscle when aimed at the neuromodulation effects.

Methods: 30 adult female Sprague Dawley rats were operated, 10 of them used as a shams control, while 10 each received double-blinded Cerebrolysin vs isotonic NaCl aqueous solution. Investigations post operatively. To guide the 1st motor neurons a stump from the sural nerve was connected from the right lateral funiculus to the distal nerve stump of internal obliquus abdominis muscle. Cerebrolysin is combination of ac-tive fragments of neurotrophic factors of lipid-free pig brain-proteins, described to have nerve protective properties. This drug was administered intraperitoneal, 5ml/kg, over a period of 14 days postoperatively. After three months post operation the rats were monitored electrophysiologically (emg, nct) for muscle re-innervation before the open local injection of the retrograde tracer fast blue. Animals were sacrificed after another 10 days for histo-neuro-pathology and immunohistochemical analysis.

Results: 1. Local functional neuromodulation was proven by an increase of GLU1 trans-porters in Western blot after three months; 2. Functional re-innervation was confirmed electro physiologically; 3. In the neurotrophic factors treated-group marked differences in the histopa-thology was shown within the spinal cord at the site of the lesions (implantation) and within the grafts that can be interpreted as neuro-protecting and neuro-restorative ef-fects: (a) reduced sprouting of Schwann cells and enhanced number of oligodendroglia into the grey and white matter around of implant were correlated with more intense axonal regeneration; (b) reduced number of astrocytes around implantation area indi-cates low atypical regeneration by cicatrisation and this feature was correlated, also, with better axonal regeneration; (c) reduced number of microglial cells and macrophag-es around graft indicate less apoptosis and less axons degeneration in spinal cord; (d) better preservation of spinal neurons beside of nerve graft; (e) reduced fibrosis and more intense axonal regeneration into the transplanted nerve.

Discussion: Functional re-activation of glutamatergic transporters in the synapses of the motor endplates dictated through peripheral nerve grafts was described by BRUNELLI et al in 2005. In addition our study showed effects of neuroprotection and neuro-restoration after acute treatment with neurotrophic factors. These effects might be crucial for restoration of motor functioning in SCI and in brachial plexus avulsions repair.

Conclusion: The neurotrophic factors medication supports CNS plasticity, neuroprotection and neuro-regeneration in rats after grafting the 1st motor neuron from the spinal cord to muscle nerve.

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TIMING OF NEUROPROTECTION AND NEUROREHABILITATION IN ACUTE TRAUMATIC BRAIN INJURIES (TBI)

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Background

Traumatic brain injury (TBI) represent a very important cause of disease in all countries. Each year TBI contribute to substantial number of deaths and cases of permanent disability. TBI is caused by a bump, blow or jolt to the head or a penetrating head injury that disrupts the normal function of the brain. The severity of a TBI may range from "mild" (a brief change in mental status or consciousness) to "severe" (an extended period of unconsciousness or amnesia after the injury).

TBI in the United States: an estimated 1.7 million people sustain a TBI annually. Of them: 52,000 die, 275,000 are hospitalized, and 1.365 million, nearly 80%, are treated and released from an emergency department. TBI by Age: children aged 0 to 4 years, older adolescents aged 15 to 19 years, and adults aged 65 years and older are most likely to sustain a TBI. Adults aged 75 years and older have the highest rates of TBI-related hospitalization and death. TBI by External Cause: Motor vehicle–traffic injury is the leading cause of TBI-related death. Rates are highest for adults aged 20 to 24 years. (U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES - Center for Injury Prevention and Control, www.cdc.gov/TraumaticBrainInjury, MARCH 2010).

TBI delimitation was performed by Teasdale and Jennett (1974), in Glasgow Coma Scale (GCS), important standard in the assessment of these brain lesions: minor (13-15), moderate (12-9), severe (8-3). This standard ("golden") scale in TBI was established by motor (1-6p.), verbal (1-5p.), eyes (1-4p.) response at external stimulus. For children (0-16 years) in all hospitals was Children Coma Scale (CCS), also quantification 3-15 points. Severe brain injuries (GCS 3-8) represent an important cause of mortality and morbidity, especially in patients with active period of live (20-40 years old).

Material & Method

I. Including criteria: the authors studied non selected consecutive 98 patients with SBI (between 6 – 66 years old), 58 male and 40 female in period 2006-2010 (5 years) at the Hospital "Bagdasar-Arseni", Bucharest. The distribution by age was children 30 cases (30.6%) and adults 58 cases (59.2%). The most frequent cause of SBI is represented by the car accidents (car to pedestrian, passenger vehicle) 58 cases (59.2%), followed by falls different higher 23 cases (24.5%) domestic accidents 4 cases (4,1%) and sport traumas 3 cases (3.1%). All intracranial hematomas were operated in the first 6 hours after admission.
Excluding criteria: all patients in SBI status with multiple traumas with or without intracranial hematomas.

All 98 cases with SBI were monitoring in intensive care unit (ICU). At admission GCS 3-4 was 28 cases (28,5%), GCS 5-6 was 29 cases (29,5%), GCS 7-8 was 41 cases (41.8%). In all cases the admission CT scan was performed immediately; The following

CT scan was performed at 6, 24, 72 hours and after 1 week to verified the brain lesion and intracranial mass lesion. In 30 cases (30.6%) intracranial mass lesions undergone to the operative procedures: extradural hematoma 14 cases (14.3%), subdural hematoma 10 cases (10.2%), intraparenchimal hematoma 6 cases (6.1%). Additional in 10 cases (10.2%) we report penetrated head injury. Also, CT scan showed hemorrhagic contusion 23/98 (23.5%) SAH in 27/98 cases (27.5%), hypodense (ischemic aria lesion in 25/98 cases (25.5%), cerebral edema 40/98 cases (40.8%) and DAI 19/98 cases (19.3%); DAI was diagnosis only by MRI and the first week post-injury. In our data surgical evacuation of mass lesions was performed as needed, but only five decompression craniotomy was done. In our study no mortality was registered in the group of ICP < 20 mmHg, all the 28/98 cases (28.5%) which died had the ICP > 20 mmHg.
In all cases, admitted in I.C.U Cerebrolisyn –as neuroprotective therapy was early administrated, in the first 24 hours. Also early neurorehabilitation represent an important therapeuticall factor in global outcome.
In the literature there are studies which correlate neuroprotective therapy with the GOS with GCS, metabolic, hematological, radiological and clinical profiles.

In our data in 98 SBI with cerebrolysin: Glasgow Outcome Scale (GOS) was good recovery was in 27 cases (27.5%), moderate disability 26 cases (26.5%), severe disability 13 cases (13.2%), vegetative state 8 cases (8.1%), death 24 cases (24.4%). At admission GCS 7-8 was preponderent 41 cases (41.8%) which it was in concordance with the global outcome. The psychological support in all SBI will be necessary to obtain social, familial and professional integration.

The control study (32 cases – GCS: 3-8pct) was realized in the same period; The GOS in control study : good recovery was in 7 cases (21.8%), moder-ate disability 5 cases (15.6%), severe disability 7 cases (21.8%), vegetative state 4 cases (12.5%), death 9 cases (28.1%).
The statistical comparative data show strong association between neurotrophic factor treatment and global outcome.(Chi-square test : p<0,001)
II. also the authors studied 26 cases with moderate brain injury (MBI), carefully selected, with special referrence of GCS score 9 points (clinical data: somnolence, neurological deficit and cranial nerve palsy; the brain stem reflex are normal). All cases were admitted in ICU. All cases received early neuroprotection therapy. GOS at discharge was good recovery 11/26 (42.3%), moderate recovery 10/26 (38.4%), severe disability 2/26 (7.6%), vegetative state 1/26 (3.8%), death 2/26 (7.6%).

Conclusions: TBI represent an important medical and neurosurgical problem. Important predictor's factors in TBI are: patient age, associated pathology and drugs, transport & hospital facilities, multiple trauma, GC Scale score at admission, CT scan abnormalities and early Neuroprotection & Neurorehabilitation. In our experience the neurotrophic factors are a significant improvement in SBI and in MBI. Cerebrolysin (mixture of low molecular polypeptide, extracted from pig brain) increase motor function, enhance the cognitive performances, increase memory & attention, improve of brain bioelectrical activity.

Keywords: traumatic brain injury (TBI), severe brain injury (SBI), moderate brain injury (MBI), GCS, DAI, ICP, neuroprotection, neurorehabilitation, outcome, GOS.

INTERVENTIONS IN ACUTE ISCHEMIC STROKE

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The re-eopening of occluded vessels improves clinical outcome in acute ischemic stroke. In iv. lysis ca. 50% of recanalisation occurs in the first hour while further re-opening (ca. 20%) might happen in the next five hours period. Re-canalization is associated with significant increase of good functional outcome and a similar reduction in the letal outcome.

The beneficial effect of early recanalisation of cerebral blood flow on stroke outcome may be negatively influenced by other factors such as extent of irreversible brain injury before re-canalization, excessive glucose level at the time of reperfusion, and blood pressure changes during thrombolysis.

The larger the clot volume the smaller is the recanalisation rate . Patients with lower amount of clot were more likely to have an independent functional outcome ($P < 0.001$). In contrast, mortality increased in patients with higher amount of clot. Furthermore, these patients were more likely to have hemorrhagic infarct transformation ($P < 0.003$) and parenchymal hematoma ($P < 0.008$) on follow-up scans. Vascular re-canalization rates vary depending on thrombus location.

Only 10% of carotid occlusions and one third of MCA occlusions could be reopened via iv. lysis. Intra-arterial thrombolysis has higher early re-canalization rate (60%) but the highest re-canalization could be achieved with mechanical devices (80%). The rate of symptomatic intracranial hemorrhages with ia. lysis is 10% vs. 2% in the placebo group. The rate of favorable outcome (mRS) is also higher than that in placebo group (40% vs. 25%). IA tPA may therefore be an option in selected patients with large vessel occlusions and limited response to IV tPA. The re-canalization rates is probably higher with IA thrombolysis than that with iv. approach, but, the clinical benefit may be reduced by the time.With combined IV and IA thrombolysis (0.6mg of IV tPA followed by IA t PA) partial or complete re-canalization was achieved in more than 60% of patients after IA treatment but only a modest trend for good outcome could be seen in clinical outcomes. Mechanical therapies are more effective in removing large thrombi in proximal vessels and associated with lower hemorrhage risk. Disadvantages are:endothelial damage and bleeding.

In a systematic review re-canalization was achieved in 70% and bleeding occurred in 20%. An interesting approach is the ultrasound enhanced thrombolysis with 2MHz ultrasound. Complete re-canalization or dramatic clinical recovery within 2h after the administration of tPA bolus occurred in 49% in the ultrasound group as compared to 30% in the control. Ultrasound enhanced lysis+microbubble combination might further increase the rate of recanalisation.

DOSE BDNF -196 G>A AND -270 C>T POLYMORPHISMS IS ASSOCIATED WITH CLINICAL COURSE OF MULTIPLE SCLEROSIS AND STROKE RECOVERY?

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Brain-derived neurotrophic factor (BDNF) belongs to the family of neurotrophins originally described to be crucial for neuronal differentiation and survival. BDNF is known to be involved in myelin formation and promote remyelination in multiple sclerosis (MS) and is also essential for post-stroke recovery in rodents. BDNF-196 G>A (Met/Val) polymorphisms has been shown to have functional consequences, whereas for -270 C>T it is suspected but was not fully elucidated. The Met allele has been associated with impairments in intracellular trafficking and activity-dependent secretion of BDNF in neurons and neurosecretory cells. We have determined impact of BDNF single nucleotide polymorphisms (SNPs): -196 G>A (Met/Val) and -270 C>T on 1) MS susceptibility and its the clinical course, and 2) human stroke recovery.

270 MS patients and 177 age- and sex matched controls were included. Expanded Disability Status Scale (EDSS) was used to evaluate neurological condition. Increased risk of MS was noted in both -196 GG and -270 CT genotypes carriers comparing to controls and earlier occurrence of the disease in BDNF -196 GG MS carriers was found.
48 stroke patients (22 with aphasia and 26 with hand paresis) were included. In aim to assess motor function and language abilities in stroke patients Fugl-Meyer, Wolf and Goodglass & Kaplan's tests were performed respectively. In both groups improvement in language and motor skills after 3 weeks training were observed, however no effect of determined BDNF polymorphisms on functional motor or language recovery was found.

Lack of effect of BDNF SNPs on clinical outcome in stroke may be explained by influence of many different genetic and non-genetic factors involved in processes of post-ischemic recovery. Further studies are needed to elucidate influence of endogenous neurotrophic factors on the rehabilitation process.

STROKE AND NEUROPLASTICITY

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Stroke is one of the leading causes of mortality and disability in modern countries. Clinical manifestation of stroke is rapidly developing loss of brain function(s) due to disturbance in the blood supply to the brain. This can be due to ischemia (lack of blood flow) caused by blockage (thrombosis, arterial embolism), or a hemorrhage (leakage of blood).

Neuroplasticity (also known as cortical mapping) challenges the idea that brain functions are fixed in certain time. It refers to ability of the human brain to change as result of one's experience, that the brain is „plastic" and „malleable". The brain consists of nerve cells (neurons) and glial cells which are interconnected, and learning may happen through change in the strength of the connections, by adding or removing connections, or adding cells. This concept is captured in the aphorism, „neurons that fire together, wire together"/"neurons that fire apart, wire apart".Neuroplasticity can act through two possible mechanisms on stroke disability-prevention and treatment of neurological deficit. A surprising consequence of neuroplasticity is used in both cases-brain activity associated with a given function can move to a different location. This is fundamental issue that supports the scientific basis for treatment of acquired brain injury with goal directed experiential therapeutic programs in the context of rehabilitation approaches to functional consequences of the injury. Same mechanism are basis for brain „fitness" in order to prevent vascular dementia or to minimize stroke injury when it happens and to prepare better basis for further neurorehabilitation if it is needed. All of these methods include modulation of NMDA receptors, 5-lypoxigenase as a controlling enzyme and cox-2 enzyme products which are involved also in pathomorfological mechanisms of atherosclerosis and stroke as well as mood disorders (depression). Common risk factors for stroke have negative influence on neuroplasticity. They are non modifiable risk factors: age, gender, race/ethnic, genotype, previous myocardial infarction, TIA or stroke and modifiable risk factors: diabetes, hyperlipidemia, arterial hypertension, atrial fibrillation, coronary and or peripheral artery disease, obesity, physical inactivity, stress, alcohol consumption, smoking. One of the important risk factor, but usually underrecognized is modern way of living, therefore we must learn how to recognize bad habits. We must learn how to cope stress with daily relaxation techniques, a personal exercise program, pertinent life style changes, a healthy diet, good sleep and appropriate nutritional habits. One of the important food ingredients which have strong impact on neuroplasticity are flavonoids, ubiquitous polyphenols in plants and vegetables, have been identified as mainly by responsible for these actions. As key regulators of cell reactivity against oxidative aggressions, the flavonoid molecule can become an ideal template for compounds therapeutically active in stroke, dementia

Some of the frequently used methods for enhancing brain plasticity are:

- Music therapy: Auditory stimulation increases mean blood flow velocity (MBFV) in the middle cerebral artery (MCA) in healthy individuals. Better circulation

enables better metabolism of the neurons and consequential neuroplasticity in both, healthy individuals and stroke patients.

- Mirror box- Due to the mirror, the patient sees a reflection of the good hand where the missing limb would be. The patient thus receives artificial visual feedback that the "resurrected" limb is now moving when they move the good hand.

- Brain fitness (multitask games) - demanding and challenging cognitive tasks engage the brain in such a way that it assimilates the new brain cells, strengthening problem solving ability.

- Brain machine interfaces with motor cortical implants are still under investigation in animal models.

BRAIN DEATH; TOWARDS AN ACTIVE DIAGNOSIS

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The diagnosis of brain death is becoming of an essential importance, specially after the erra of organ transplantation. Brain death is irreversible cessation of all clinical functions of the brain. Three prerequesites should be fulfilled first: (1) It is absolutely certain that irremidiable brain damage has occurred due to a known irreversible cause (2) A patient is not dead untill he is warm and dead (3) Exclusion of intoxication. Only when these 3 prerequesites are fulfilled, should one proceed with a clinical examination to determine brain death, which includes 3 cardinal findings: (1) Coma (2) Absence of brainstem reflexes (3) Apnea. Confirmatory tests are recommended only when the proximate cause of coma is not known or when confounding clinical conditions limit the clinical examination. These confirmatory tests include: EEG, cerebral angiography, transcranial ultrasonography, cerebral scintigraphy, brainstem evoked potentials, MRI, MRA, MRS or PET. The srandards are variable between countries and even between different states in USA.

THE EFFECT OF NEUROPROTECTION AND REPETITIVE TRANSMAGNETIC STIMULATION IN STROKE PATIENTS.

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Introduction: Stroke is one of the primary causes of death and disability in the adult population.Studies indicate that as many as 50% of survivors of a stroke remain permenantly disabled (Goldstein etal 2001.)Recurrence of stroke is common and is a major contributor to the disability and mortality of the disease.The risk of recurrence is highest in the first month following a cerebrovascular event and although it then subsequently falls,around 20% of survivors will have another stroke within 5 years(Hankey 2003,Sacco 1998).Ischemic stroke is a heterogeneous disorder generally caused by one of three pathogenetic mechanisms:atherosclerostic disease in extracranial and large intracranial arteries;embolism from the heart and intracranial small vessel disease. (Sacco,1998).Asian,African and Hispanic populations have significantly higher rates of intracranial stenotic disease than North American Caucasians and it has been consistently observed that this places them at an increased risk for stroke,heart disease and death.(Chimowitz etal,1995). Preliminary studies on Egyptian patients have also shown comparable high rates of intracranial stenotic disease(Zaki-eldine 2002).Less than 1% of the acute stroke patients have access to tpa and thrombolysis in the first 3-6 hours after stroke in Egypt.Strategies towards management of the after stroke use antiplatelets or anticoagulants plus antilipids to reduce the risk of recurrence plus control of the underlying risk factors and physiotherapy.The Aim of the work in this study is to test the value of adding neuroprotection plus repetitive T.M.S. to the standard management of after stroke victims.The results of adding neuroprotection plus repetitive Transmagnetic Stimulation will be discussed.

BRAIN REPAIR THERAPIES IN ISCHEMIC STROKE. THE ROLE OF TROPHIC FACTORS AND STEM CELLS

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Brain repair is an endogenous natural mechanism that is activated following stroke. From a therapeutic point of view it is desirable to find repair therapies that increase the possibilities of recovery by encouraging cerebral plasticity. We review the bases of brain repair in ischemic stroke, particularly in the therapeutic administration of trophic factors and stem cells.

Trophic factor exogenous administration or endogenous stimulation of the same in animal models of cerebral infarction has shown certain efficacy in increasing neurogenesis and angiogenesis, enhancing functional recovery, and in reducing infarct volume and neuronal cell death. Three trophic factors have been tested in stroke clinical trials: A phase III trial of bFGF was stopped due to side effects; EPO and G-CSF are still under investigation. CDP-choline (citicoline™) and porcine brain derived peptide are drugs that could act stimulating trophic factors and could contribute to a repair effect (decreasing apoptosis and increasing neurogenesis, synaptogenesis and VEGF) as suggested by the results obtained in animals models. Clinical trials with CDP-choline, drugs with trophic-factor effect, showed safety. At the moment, there are phase III clinical trials with those drugs ongoing.

Stem cell administration in animal models has shown efficacy in promoting functional recovery, neurogenesis and gliagenesis, reducing infarct volume and has anti-apoptotic activity. Clinical trials showed safety and feasibility in phase I and phase II with neuronal stem cells and bone marrow derived mesenchymal stem cells. Studies in phase III are needed to assess the effect of stem cells on stroke outcome.

Brain plasticity is an endogenous process that can be enhanced by administration of trophic factors and also by the exogenous administration of stem cells.

MITOCHONDRIAL FUNCTIONS AND DYSFUNCTIONS IN SOME INHERITED NEUROLOGICAL DISEASES AS A MODEL OF NEURODEGENERATION AND NEUROPROTECTION

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Mitochondria are well known cell organelles related to energy production , particularly important in the tissues with high energy request as nervous system and muscle. Mitochondrial dysfunctions have been recognized in a series of neurological disorders, in relationship to impairment of mitochondrial DNA and nuclear DNA and a series of syndromes called mitochondrial encephaloneuromyopathies have been described.
All are mainly clinically characterized by multisystem involvement, including brain, eye, endocrine system, muscle, hearth, nerves, etc. and some syndromes have been defined as MERRF (Mitochondrial encephalopathies with ragged red fibres), MELAS (Mitochondrial Encephalopathy Lactic acidosis and stroke-like episodes), Kearns Sayre syndrome, NARRP, etc.

Patients with disorders from mutations in the mitochondrial genome have variable phenotypes, but common to many of these disorders are underlying changes in postmitotic cells, particularly neurons and muscle fibers. The mitochondrial dysfunction caused by these mutations has been shown to be associated with signs of apoptosis and to cause cell loss.

Mutations of the mitochondrial genome have also been shown to accumulate with age and in common neurodegenerative diseases, such as Parkinson's disease.

Here, we will also discuss the potential role of mitochondrial fission and fusion in the onset and progression of neurodegenerative diseases. Specifically, an imbalance in mitochondrial fission and fusion may be the basis of familial and sporadic neurodegenerative disorders. First, hereditary mutations in the mitochondrial fusion GTPases optic atrophy-1 and mitofusin-2 cause neuropathies in humans. In addition, recent findings report increased mitochondrial fission in Parkinson's disease (PD) models and induction of mitochondrial fission by two proteins, PTEN-induced kinase 1 and parkin, which are mutant in familial forms of PD and other diseases.

We will describe

-our experience in the diagnosis of several mitochondrial disorders as a model of energy impairment neurodegeneration
- our research on oxidative stress induced apoptosis in several neurometabolic and genetic neurodegenerative diseases.
- some therapeutic approaches discussing the potential therapeutic efficacy of creatine, coenzyme Q10, idebenone, synthetic triterpenoids, and mitochondrial targeted antioxidants (MitoQ) .
This review presents recent data to show that the information gained from studying patients with mitochondrial disorders can help our understanding of the role of mitochondrial DNA mutations in brain aging and generally in neurodegeneration.

COMPLEX INVESTIGATION OF ANGIOGENESIS IN GLIOBLASTOMA MULTIFORME STEM CELLS

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Objective:
Angiogenesis is an important prognostic factor associated with tumor growth and progression. Starting from personal clinical observations in some cases of re-intervention for recurrence in patients treated with temozolomide (TMZ), regarding to the development of particular aspects of angiogenesis, specific tumor angiogenic characteristics were investigated, to identify the most suitable prognostic factor in generating multimodal therapy protocols.

Material and methods: Fresh tumor biopsies were processed for obtaining established cell lines and for development of a three dimensional (3D) functional angiogenesis assay in fibrin-gel matrix supplemented with serum-free growth medium, in presence of TMZ and anti-angiogenic or pro-angiogenic factors (bevacizumab, sunitinib, VEGF, EGF and PDGF). Chemosensitivity for TMZ of isolated tumor cells was determined by MTT test. Genes implicated in angiogenesis were evaluated at mRNA level by real-time PCR in tumor tissue and peri-tumoral tissue as control: VEGF, PDGF, TNF-ß, ICAMs, CTGF, EPCR. Microvascular density (MVD) was also determined using a protocol adapted after Weidner's method

Results: In 14 established cell lines, tumor cells tested by MTT assay shown sensitivity for TMZ in 60% of cases. Spontaneous angiogenesis was observed in most of the tumor explants tested and the development and spreading of capillary structures were enhanced in presence of TMZ in 11 from 19 cases (57.8%). MVD values varied between 22-130, with a median value of 83.6. Enhanced angiogenic potential in 3D model was correlated in 4 cases with higher MDV results (MDV>90) (21%) and in 5 cases with an increased expression of pro-angiogenic genes(26%). Higher levels of VEGF and PDGF mRNA were observed in 47% of tumors.

Conclusions:
A great individual variability was observed in tumor cells sensitivity to TMZ and in angiogenic potential of GM, suggesting that these processes are controlled by multiple factors, mainly by the presence of growth factors such as VEGF and PDGF, but in correlation with other variable local factors from the vascular niche. TMZ enhanced angiogenesis in some tumors, probably by selection of cancer stem cells. A complex evaluation of each tumor can indicate the best choice of further therapy.

NEUROTROPHIC FACTOR IN TREATMENT OF CONSCIOUSNESS DISTURBANCES IN ICU AND ITS INFLUENCE ON BRAIN TISSUE OXYGENATION MEASURED WITH NIRS MONITOR.

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Consciousness disturbances in ICU are met in about 15% of admitted patients. Those disturbances can be divided into two subgroups: quantity (ie. coma, semicoma) and quality (ie. dementia, delirium, cognitive disturbances). The definition of coma stays that this is a state similar to sleep but without self-consciousness and place-time orientation even after strong stimulation. There are some diseases simulating coma ie. lock-in syndrome, severe brain injury, vegetative state, brain death, profound dementia. The comas can be divided into structural (ie. brain trauma, brain tumors) and metabolic (functional ie. intoxication, infection, drug overdose, metabolic disorders, hypothermia). Every pathological mechanism leading to diffused brain cortex destruction can cause coma including drop in blood pressure, brain contusion etc. The outcome prognosis in post-brain injury comas depend on several factors but generally is better comparing to non-post-traumatic comas (death in 11% vs 88% within 12 months) [1,2]. If patient in non-post-traumatic coma (ie. after cardiac arrest) presents no reaction in evaluation of brain steam reflexes after 3 days of treatment the risk of death is very high and can reach 97% within 2 months [1,2]. In post-traumatic comas the factors influencing negatively outcome are intracranial haematoma, low GCS and high ISS score at admission to ICU. The age over 65 yrs is independent factor in both groups. The mechanisms of brain cells – neurons injury is complicated and includes hipoperfusion, acidosis, cytotoxic brain oedema. This involves many complicated pathology mechanisms at level of cell metabolism and as a consequence leads to neurons death and profound encephalopathy. Few years ago the possibility of neurogenesis in adults was discovered. The stem cells were found in central nervous system of grownups. The neurogenesis process consist of 3 steps: non-symetric division of cells, migration of new cells – potential neurons to some part of the brain, and differentiation and integration at place. This process is physiological in some parts of brain and can be stimulated by some chemical substances. This new, alternative method of treatment of patients after severe brain-injury can be effective and lead to improvement of patients outcome including increasing the quality of life. So far several substances were used and tested: nimodypina, piracetam, amantadyna and cerebrolysin. The cerebrolysin is promising drug in treatment of brain function disturbances caused by brain injury. The cerebrolysin is a mixture of low molecule peptides which have neurotrophic potential. This drug stimulates the neurogenesis and interferes with pathological mechanisms in post-traumatic brain cells reducing necrosis and apoptosis of neurons. Its mechanism of action is well described in many papers [3-10]. In observational study we decided to evaluate brain blood perfusion and brain tissue oxygenation in patients receiving neurotrophic factor treatment after brain injury. The brain tissue oxygenation was assessed with NIRS (near infra-red spectroscopy) monitor. The electrodes were placed on cranium of the patient and tissue oxygenation monitored in time intervals. This monitoring is non-invasive method and does not interfere with patient`s treatment.

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NEUROINFLAMMATION AND NEURODEGENERATION IN MULTIPLE SCLEROSIS AND ITS EXPERIMENTAL MODELS

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In the last several years neurodegeneration has been rediscovered in multiple sclerosis (MS). Mechanisms leading to development of axonal loss in this disease which is characterized by prominent inflammation and demyelination in the central nervous system (CNS) are still poorly understood. Understanding of this mechanism would be very important from the clinical point of view because neurodegeneration is generally accepted as the main determinant of permanent clinical disability. Relationship between neuroinflammation and neurodegeneration in MS is complicated. There are some studies suggesting that inflammation in the CNS may induce axonal loss. Some inflammatory cells directly or through secreted proinflammatory mediators may initiate damage to myelin and axons. In contrary, some other results show that neurodegeneration initiates inflammation. Neurons can produce inflammatory cytokines and stimulate surrounding glial cells to express MHC class I and II molecules. There are also some suggestions that inflammation can protect against neurodegeneration. Several experimental in vivo and in vitro models has been used to simplify the complex picture of MS pathology and to study the relationship between neuroinflammation and axonal loss in this disease.

COMPLEX INVESTIGATION OF ANGIOGENESIS IN GLIOBLASTOMA MULTIFORME STEM CELLS

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Most Alzheimer pathology and cerebral infarcts show no clinical symptoms. Hence, if we are to prevent stroke and delay Alzheimer disease, we need to begin much earlier.

The molecular mischief leading to Alzheimer's disease probably begins about two decades before the first clinical manifestations. For each clinical stroke, there are five or ten subclinical strokes that do not have symptoms, but on closer examination, often have subtle signs, most frequently abnormalities of executive function. Epidemiological, clinical and experimental evidence suggest that stroke and Alzheimer disease may interact, at least in a subset of patients.

Stroke and Alzheimer disease share the same treatable risk factors and consequently, treatment should begin early and vigorously. Trials need to be done, not only using new strategies, but with a specific aim of preventing stroke and delaying Alzheimer's disease. It is never too late to intervene, but the earlier, the better. Prevention at the pre-symptomatic stage may be the new frontier in the prevention of the two greatest threats to the brain, stroke and Alzheimer's disease.

SUBTYPES OF NONCONVULSIVE STATUS EPILEPTICUS

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In recent years, various alternative classifications of nonconvulsive status epilepticus (NCSE) have been suggested, which has loosened the classification from the constraints of a purely seizure type schema (Shorvon,1994,Walker et al 2005). There are different subtypes of NCSE in epileptic patients. NCSE is een in the neonatal and infantile epilepsy syndromes, childhood, adults with and without encephalopathy. However there is an interesting and controversial aspect associated with NCSE.This entity of NCSE is seen in some cases of encephalopathy, acute traumatic head injuries in coma and metabolic confusional states. EEG is mandatory to detect electrographic activity to diagnose NCSE. The EEG exhibits continuous or periodic EEG abnormalities. The natural history of NCSE is not known.However, even if NCSE doesnot cause death,it could still cause neuronal injury.If this is true, then aggressive therapy may be warranted to prevent neuronal injury and possible long term sequelae especially epilepsy and encephalopathy (Shneker and Fountain,2003)

FUTURE ASPECTS OF MOTOR THERAPIES IN NEUROREHABILITATION

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Within the last decade there was a dramatic change in paradigms in motor rehabilitation: Physiotherapy is no longer understood as “hands on” treatment but concentrates more on “hands off” and coaching activities. The traditional “school” oriented concepts are more and more replaced by therapeutic procedures which are derived from neurobiological and neurobehavioural knowledge and are evidence based.

To further stimulate progress in the field of motor rehabilitation a fast transfer from basic neuro- and behavioural sciences into clinical practice is needed and appropriate clinical study designs and service implementations have to be defined.

Several evidence based therapeutic procedures can be grouped into modules to ascertain that every patient has a chance to be treated by a procedure likely to improve his condition even on a limited length of stay. So a quality proven rehabilitative therapy can be offered.

In the talk both neuroscientific principles of plasticity and motor learning as well as examples of therapeutic modules will be demonstrated . Furthermore the now most promising and innovative concepts based on the mirror neuron system and motor learning by imagery and imitation will be introduced and discussed.

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WHY HAVE WE FAILED TO CURE ALZHEIMER'S DISEASE?

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There is widespread recognition in the urgency to understand the causes and mechanisms of senile dementia. Attempts to find cures for Alzheimer's disease (AD) have, however, failed so far, in spite of enormous investments, intellectual and financial. We therefore have to reconsider the problem from new angles. AD is regarded as a disease because of its clinical manifestations and underlying pathology. However, this combination does not define a disease but rather a syndrome, just like hepatic cirrhosis in which liver pathology causes metabolic changes, but which can result from many different etiologies. It is unlikely that attacking a downstream phenomenon, like apoptosis or ß-amyloid accumulation, can cure AD, or prevent the progression of the disease.

It is probable that senile dementia is the result of a combination of several processes, working differently in each person. Epidemiological studies have identified many risk factors for "senile dementia of the Alzheimer type", some genetic but most environmental and therefore modifiable. Therefore a concerted action to fight the dementia epidemic must be made by aggressive action against its risk factors, and this battle must begin in midlife, not in old age.

ATTENTIONAL MECHANISMS OF MOOD CONTROL: NEUROPHYSIOLOGICAL CORRELATES OF MINDFULNESS IN RECURRENT DEPRESSION

Experimental studies of attention are largely concentrated on the effects of external distraction. In contrast, a big cognitive problem in depression is the internal distraction in form of rumination. The ruminating cognitive set is an important mediating factor, which is present during remission and plays a central role in the transition from remission to relapse. Since mindfulness is a cognitive set opposite to rumination, mindfulness training has been applied for prophylactics of relapses in chronic depression. The aim of the present study was an analysis of neurocognitive mechanisms of this training. Forty patients with three major depressive episodes in the past (presently in remission) were presented an eight-week mindfulness training course. Slow negative cortical potentials (SCP) as indicators of the allocation of attentional resources were recorded during passive stimulation before and after training. The two control groups were healthy individuals and matched recurrent depressive patients. Mindfulness training resulted in a significant and highly reliable (split-half analysis) increase of the SCP amplitude. In contrast, both control groups revealed a trend to SCP decrease. The data indicate that mindfulness training leads to the suppression of interfering thoughts in depressive patients, and that this suppression of inner interference results in freeing cognitive resources for processing of environmental stimuli.

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RECENT ADVANCES IN NEUROREHABILITATION.

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Summary. Modern neurorehabilitation strategies are based in neuroscience. Rehabilitation intervention can result in structural reorganization in damaged human brains and the magnitude of this structural change is directly proportional to the amount of clinical improvement. Use-dependent plasticity of the central nervous system is attributed to both dose-dependent and context dependent effects of rehabilitative intervention referred as enriched environment and enriched rehabilitation. Plastic structural brain changes can be produced by different motor therapies. Constrain-Induced Movement Therapy and Modified Constrain-Induced Movement Therapy combined with clinical problem-solving process remain a promising motor intervention however its efficacy appears to be enhanced by use of mental practice provided directly after CIMT clinical sessions. Technological advances continue to inspire the specialists of neurorehabilitation with both excitement and apprehension. A challenge for clinicians is to determine which of the growing number of devices or interventions available should be incorporated into their clinical practice, and when and with whom they should be offered, in order to best help their patients in attaining the highest level of function and quality of life.

Key words: neurorehabilitation, advances

NEURAL CORRELATES OF EMPATHY TO PAIN IN PATIENTS WITH DISORDERS OF CONSCIOUSNESS

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Introduction: Pain and suffering controversies in persons with disorders of consciousness continue to be debated by the scientific and medical ethics communities. Previous studies have shown contradictory results regarding pain and suffer in vegetative state patients. Thus, data to date do not allow for differentiation of the degree of any conscious pain experience of whether individuals with disorders of consciousness are able to suffer.

Methods: Using functional magnetic resonance tomography (fMRT), empathy to pain and suffer was assessed in 22 healthy subjects, 3 minimally conscious patients (MCS), and 5 persistent vegetative patients (PVS). The stimuli consisted of 10 pain-related exclamations and 10 non-painful exclamations, which were presented via headphones.

Results: Compared to the control condition, healthy subjects showed enhanced activation to pain-related exclamations in the left insula, superior and middle temporal gyri, and cerebellum. Two PVS patients showed greater activation in the superior temporal gyrus and insula, one MCS patient showed greater activation in the supratemporal gyrus, insula, amygdala, and inferior frontal gyrus, and one MCS patient showed more activation in the left superior temporal gyrus.

Conclusions: The findings indicate that in some PVS and MCS patients similar pain-related responses are present as in healthy controls.

NEUROPLASTICITY AND BRAIN REPAIR AFTER STROKE

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Stroke is a leading cause of discapacity. Spontaneous recovery occurred in the days, weeks and even months after stroke. Constantly, new insights emerge into the neurobiology of repair after stroke in animals as well as in humans, going from molecular, vascular, glial, neuronal, to behavioral and environmental events. All together they are setting the grounds for new therapies for improving brain repair after stroke. The ability to image the events that occur while neuroplasticity takes place in the brain -not only during spontaneous but most importantly during rehabilitation interventions- is encouraging. The information being gathered so far, is clearing the road as to define specific interventions that promotes brain recovery

NEONATAL KETAMINE ANESTHESIA, NEURONAL CELL DEATH AND LONG-LASTING COGNITIVE DEFICITS IN NONHUMAN PRIMATES.

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Our laboratory has shown in the rhesus monkey that 24 hours of clinically relevant ketamine-induced anesthesia causes significant increases in neuronal cell death and that the sensitivity to this ketamine effect is apparent as early as gestational day 122 (in utero exposure via maternal anesthesia) and as late as postnatal day (PND) 6, but is not seen on PND 35. For our functional assessments, six monkeys were exposed on PND 5 or 6 to intravenous ketamine anesthesia (light surgical plane) for 24 hrs; six control animals were unexposed. At 7 months of age all animals were weaned and began training to perform a series of cognitive function tasks as part of the National Center for Toxicological Research (NCTR) Operant Test Battery (OTB). The OTB tasks used here included those for assessing aspects of learning, motivation, color discrimination, and short-term memory. Subjects responded for food (banana-flavored pellets) by pressing response levers and press-plates during daily (M-F) test sessions (50 min) and were assigned competency scores based upon their individual task performance. Beginning around 10 months of age, control animals outperformed (had higher training scores than) ketamine-exposed animals for approximately the next 30 months. For these animals that now over 4 years of age, the cognitive impairments continue to manifest in the ketamine-exposed group in the OTB learning task as deficits in accuracy and decreases in rate of responding. There are also apparent differences in levels of motivation to work for food in these animals and this may be impacting their OTB performance. Decreased accuracy of performance in the color discrimination task, while lasting approximately 10 months, is now no different from that of controls; however, response rate for this task remains lower than that of controls. These observations provide further evidence that a single 24-hr episode of ketamine anesthesia, occurring during a sensitive period of brain development, results in very long-lasting deficits in brain function in primates that are not just a result of developmental delay. Supported by NICHD, CDER/FDA and NCTR/FDA.

PATHOLOGICAL NEUROPLASTICITY IN IMPULSE CONTROL DISORDERS

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Impulse Control Disorders (ICDs) are grouped as a heterogeneous cluster of disorders characterised by the "failure to resist" impulses to engage in harmful, disturbing or distressing behaviours.

ICDs include pathological gambling (PG), kleptomania, pyromania, intermittent explosive disorder, trichotillomania and ICD not otherwise specified. Criteria for other ICDs have been proposed including compulsive shopping, problematic computer use, compulsive sexual behaviour, and compulsive skin picking. Some disorders, such as substance use disorders, Tourette's syndrome, Attention Deficit Hyperactivity Disorder are characterized by impaired impulse control.

Others psychiatric disorders or behaviors that share features of ICDs have been reported to occur in Parkinson 's disease (PD) in the context of dopamine replacement therapy. PG is the most extensively studied ICD in PD.

Due to the multiple similarities between ICDs and substance addiction (in natural history, phenomenology, neurobiology, genetic), many investigators proposed that the ICDs should be best aligning with behavioral addictions.

Multiple brain regions and neurotransmitter systems (e.g., dopaminergic, noradrenergic, serotonergic, opioidergic) contribute to impulsive behaviours. The crucial neural network seems to be the cortico-striato-thalamo-cortical pathway.

This presentations aims at describe the neurocircuitry, as well as mechanisms of pathological neuroplasticity that lie beneath the adaptive behavioral responses to motivationally salient events, which is the substrate of ICDs.

OUR EXPERIENCE CONCERNING NEUROREHABILITATIVE OUTCOME IN POST-TRAUMATIC BRAIN INJURY PATIENTS, TREATED WITH NEUROTROPHIC FACTORS

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Objective: To assess neuroreahabilitative outcome, obtained in our NeuroRehabilitation clinic division, during the last 6 years, in post-traumatic brain injury (TBI) inpatients treated with neurotrophic factor, compared to inpatients who did not receive this multimodal acting drug (all during their first admission).
Study design: Comparative retrospective analysis between Cerebrolysin (different doses: 5 ml,10 ml or 20 ml per day, respectively total quantities – ml: min 25, max 610, average 174, med 140 - related to durations of administration – days: min 4, max 61, average 17.79, median 14), vs. patients non-treated with Cerebrolysin (all the inpatients received aside, a rather equivalent pharmacological and physical therapy).
Material and Methods: There have been studied two lots of patients, admitted during 2005 - 2010: 54 treated with neurotrophic factor (15 F, 39 M; mean age: 34.46 years old, me-dian 32.5) and respectively, 67 – controls – without (12 F, 55 M; mean age: 39.74 years old, median 38).
The total number of assessed items was 12: admission and discharge Functional Independence Measure (a FIM, d FIM), number of days till the first: functional knee extension (KE), walk between parallel bars recovery (WPB), cane assisted walk recovery (CWR), independent walk recovery (IWR), stairs ascent/ descend recovery (SR) and respectively, Glasgow Outcome Score (GOS) and Rankin Disability Score (RDS) - each of them at: admission (GOS_01, RDS_01), 10 days after (GOS_10, RDS_10) and 30 days after (GOS_30, RDS_30) -, number of hospitalization days (H) age (A), Glasgow Coma Scale (GCS).
Statistical analysis entailed differentiation methods after studied populations' normality evaluation of distribution, with parametrical (T) test or non-parametrical (CHI2) test and multiple linear regression to identify explicitation level of the dependent variable d FIM by the afore mentioned independent variables, within a predictive formula. The soft used were Statistical Package for Social Sciences (SPSS) and respectively Epi info.
Results and Discussion:
The dependent variable d FIM has been explicited, by their contributivity, through: a FIM (p=0.000), IWR (p=0.000), WPB (p=0.010), GOS_01 (p=0.019), age (p=0.026), with the regression coefficient R2= 0.871 (the rest of the assessed independent variables did not meet significant values).
For the neurotrophic factor-treated lot, the average of the difference between d FIM and a FIM was sig-nificantly bigger - i.e. better - for neurotrophic factor administrated in quantities of less than 120 ml/cure (Av 43.32, St dev 33.65, p<0,0001) and even better for neurotrophic factor ad-ministrated in quantities of more than 120 ml/ cure (Av 52.03, St dev 32.88, p<0,0001) - recovering a much greater amount of dysfunction - compared to the controls (Av 25.08, St dev. 29.99),
Conclusion: The neurotrophic factor treatment proved, on a rather long time extended experience of its use in our clinic division, to bring statistically significant contributions to the improvement of the neurorehabilitative outcome in post TBI patients.
Key words: traumatic brain injury, multimodal acting, neurohabilitation

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NOVEL METHODS OF POST-STROKE REHABILITATION ENHANCING USE-DEPENDENT PLASTICITY OF BRAIN

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According to literature the post-stroke patients are most numerous group of clients needing rehabilitation as for neurological diseases. One of the most importing tasks in improving arm and walking function. The latest reports on contemporary rehabilitation after stroke discribing Constraint-Induced Movement Therapy, transcranial magnetic stimulation, electrical stimulation, motor imaginery, virtual reality (VR), robot assisted therapy systems (exoskeletons), regional anaesthesia of forearm or arm and many others opened new perspectives for re-education and improving function following stroke. Most of modern exercises enhance use-dependent plasticity of brain.

Henderson et al. wrote in 2008 that the current evidence on the effectiveness of using VR in the rehabilitation of the upper limb in patients with stroke is limited but sufficiently encouraging to justify additional clinical trials in this population (Top Stroke Rehabil. 2007;14(2):52-61). Other reports on VR: Broeren et al. Stud Health Technol Inform. 2008;136:77-82, Crosbie et al. Disabil Rehabil. 2007; 29(14):1139-1146, Deutsch et al. Stroke. 2006;37(6):1477-1482, Merians et al. Neurorehabil. Neural Repair 2006; 20(2): 252-267, Schetino et al. Exp. Brain Res. 2003; 151(2): 158-166, You et al. Stroke. 2005; 36(6):1166-1171.

Bjorkman et al. studied in 2004 the effects on hand function of right forearm anaesthesia using a local anaesthetic cream (Eur. J. Neurosci. 2004; 20(10): 2733-2736). Their findings opened new perspectives for sensory re-education and rehabilitation following injury to the peripheral and central nervous system. Muelbacher et al. described in 2002 a novel therapeutic strategy (deafferentation produced by a new technique of regional anesthesia of the upper arm) that may help improve hand function in patients with long-term weakness after stroke (Arch Neurol. 2002; 59(8): 1278-1282).

New robotic solutions improve the efficiency of therapy treatments because the exercises are self-initiated, self-directed, functional and intense. Even severely impaired patients can practice independently, without the constant presence of a therapist, allowing patients to exploit their full potential for recovery. The augmented feedback provided by the shared software platform, encourages and motivates patients to achieve a higher number of repetitions, and this leads to better, faster results and improved long-term outcomes. The software also provides automatic, ongoing assessment of motor functions and patients can readily track their progress, helping them to grasp the initiative and reach towards recovery. The rehabilitation, from immediate post-injury to long-term recovery, referred to here as the "Continuum of Rehabilitation", requires a range of therapies to address the changing needs of the recovering patient (Johnson G. et al. Proc. Inst. Mech. Eng. [H]. 2001; 215(3): 275-284, Kwakkel G. et al. Neurorehabil Neural Repair. 2008; 22(2): 111-121).

One of the latest methods is Treadmill Training with Partial Body Weight Support, shortly BWS (McCain et al. Arch Phys Med Rehabil. 2008; 89(4): 684-691.

EARLY NEUROTROPHIC FACTOR TREATMENT IN TRAUMATIC BRAIN INJURY – A LARGE RETROSPECTIVE, NATIONAL, MULTICENTER COHORT STUDY

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In this study were included 7769 adult patients with traumatic brain injury (TBI), admitted in 10 departments of Neurosurgery in Romania, between 2005 -2010. Patients were managed according to the guidelines, part of them (1618 patients) receiving neurotrophic factors add-on treatment, started in the first 48 hours after TBI. Exclusion criteria were: life-threatening multiple trauma, severe other associated conditions, epilepsy, concomitant stroke, pregnancy or lactation or other concomitant medication with neuroprotective or nootropic effects, except Cere-brolysin. At baseline, all patients were evaluated according to diagnosis guidelines, fol-lowing a unique protocol in all 10 centers. From the medical records, general data were collected at admission (gender, age, etiology, medical history, concomitant medica-tion, Glasgow Coma Scale score, clinical neurological examination, CT result, whether a surgical intervention was performed) and at days 10 and 30 post-TBI patients were ranked on Glasgow Outcome Scale (GOS) and Modified Rankin Disability Score (RDS). The safety assessments included adverse events, vital signs, laboratory tests and clini-cal examinations, extracted from the patient medical records. The primary objective of this study was to test the outcome in neurotrophic factor treated patients compared to the control group, at 10 and 30 days post-TBI, and the secondary objective to evaluate the safety for TBI patients. The neurotrophic factors treated patients were separated in 2 groups, according to 2 different drug regimens (20 ml or 30 ml/day), and compared to the control group. Statistical comparison was carried out based on the stratifica-tion of patients in subgroups, depending on GCS scores at admission (severe, moderate or mild TBI). In mild TBI, patients treated with neurotrophic factors had significantly higher GOS and lower RDS scores at 10 days post-TBI, but not at 30 days post-TBI (probably due to a 'ceiling effect'), as compared to control. In moderate and severe TBI, patients treated with neurotrophic factor had significantly higher GOS and lower RDS scores both at 10 days and 30 days post-TBI, as compared to control. Moreover, in all TBI patient population, we found a significant correlation between the neurotrophic factor treatment and the prognosis at both 10 and 30 days post-TBI. In conclusion, this large retrospective study shows significant beneficial ef-fects on outcome of early neurotrophic factor treatment in TBI.

ELECTROPHYSIOLOGICAL DISFUNCTION OF CORPUS CALLOSUM IN PATIENTS WITH MULTIPLE SCLEROSIS

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Background: Multiple sclerosis(MS) is an idiopathic inflammatory demyelinating disorder of the central nervous system characterized by demyelination and axonal degeneration. Corpus callosum(CC) is commonly involved during the disease process leading to atrophy (93%). The aim of our study was to determine frequency of motor and transcallosal conduction abnormalities at MS patients, the relationship between disability of pyramidal functions and transcallosal conduction abnormalities and to determine if transcallosal inhibition investigation increase sensitivity of transcranial magnetic stimulation(TMS) in detecting central conduction deficits in MS.

Materials And Methods: Thirty six patients with clinically definite multiple sclerosis and eighteen volunteers were evaluated. TMS parameters were evaluated using single pulse TMS and a figure of eight coil. Central motor conduction time(CMCT), latency(LTI) and duration(DTI) of callosal inhibition were recorded.

Results: All TMS parameters were obtained in healthy volunteers. In MS group TI was not obtain unilaterally in 7 patients (22,6%) and bilaterally in 4 patients (13%). CMCT was abnormal in 80%, LTI in 77.4% and DTI in 93.5% of patients. A significant correlation was found between the magnitude of pyramidal disability and degree of impairment of callosal conduction parameters.

Conclusions: The high incidence of transcallosal conduction abnormalities proves the significant involvement of corpus callosum in demyelinating processes in MS. These abnormalities are more sensitive in comparison with CMCT alterations. TMS investigation should also contain transcallosal inhibition examination in order to reach a higher sensitivity of abnormalities detection in MS patients.

RISK FACTORS FOR COGNITIVE DECLINE AND DEMENTIA

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There is epidemiological evidence that diabetes type II is a potential risk factor for Alzheimer's Disease (AD). This assumption is strengthened by experimental data from icv streptozotocin-treated rats that cognitive decline is evident already two weeks after such application while molecular changes are significant only beginning after 3 month. They correspond to an insulin-resistent brain state and are similar if not identical to changes of the insulin/insulin-receptor cascade in AD. All this contrasts to Parkinson's Disease (PD) for which there is a weak correlation to diabetes type II only.

Depression, however, is a well-known risk factor for both AD and PD. It is unknown, which subtypes of depression preceed AD and PD. As a working hypothesis we assume that depressed patients who respond to a dexamethasone suppression test with increased cortisol release are proned to rather develop AD later in life while depression preceeding PD is due to degeneration of locus ceruleus and raphe neurons in the early and asymptomatic phase of PD. Such assumption has to be verified or falsified in clinical studies.

NEUROPROTECTIVE THERAPEUTIC RESULTS IN COMATOUS PATIENTS WITH AMANTADIN SULFATE

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Objective: Controlling excitotoxicity is the key to efficient neuroprotection in all acute and chronic brain injury. The classic strategy in neuroprotection is to suppress pathophysiological processes. Oxidative stress involves the generation of reactive species, among them reactive oxygen species (ROS). ROS induce both lipid peroxidation and lipid-indepent modifications of membrane proteins and can alter physical and functional properties of neuronal membrane.

Patients And Metod: Patient group consists of all patients with severe head trauma (GCS<8) admitted to the ICU of the KAIM in JMF and UHM between January 1st 1999 and April 30th 2000. The patients were divided into two groups based on the fact, whether they did or did not receive amantadin sulfate during treatment course. Group 1 consisted of 15 patients with average age 51.6 ± 16.9 years, 13 of whom were men (average age 52 ± 16.8 years) and of 2 women (average age 49 ± 24 years). Group 2 included 28 patients with average age 44.8 ± 19.1 years consisting of 24 men (average age 44.7 ± 17.7 years) and 4 women (average age 45.5 ± 30.2 years). Glasgow Coma Scale (GCS) on admission (post- resuscitation) and on discharge from the ICU were recorded and compared. Both groups were treated with the standard therapy of severe head injury accepted in our institution. On addition, group 2 patients received amantadin sulfate in the dose of 200 mg i.v. twice daily for 3 days. The persistent coma indicated of amantadin sulfate.

Results: In the group 1 the average income GCS was 5.2 ± 1.6 and the average outcome GCS was 12.4 ± 1.1. The average hospitalisation length was 8.4 days. In the group 2 the average income GCS was 4.8 ± 2.2 and the average outcome GCS was 9.9 ± 4.2. The average hospitalisation range was 9.4 days. By using the t-test, the outcome GCS in the group 2 was significantly higher than the outcome GCS in the group 1 ($p < 0.05$).

Conclusion: We found, that outcome GCS in patients with severe brain injury treated together with standard therapy and amantadin sulfate was significantly higher than in patients treated by standard therapy only. The protective effect of amantadin sulfate could be based on its blocking the brain NMDA postsynaptic receptors. This blockage of the neurons in reticular formation and allocortex can, in an optimal dose, cause desinhibition and arousal.

MINDFUL AWARENESS, QUANTUM MECHANICS AND SELF-DIRECTED NEUROPLASTICITY

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Neurobiological research generally assumes that brain mechanisms alone will ultimately suffice to explain all psychologically described phenomena. This assumption stems from the idea that all causal mechanisms relevant to neuroscience can be formulated solely in terms of the principles of classic Newtonian physics. Thus, terms having intrinsic experiential content (e.g. 'feeling', 'observing' and 'effort') are not included as primary causal factors. This theoretical perspective is dictated by ideas about the natural world that have been known to be fundamentally incorrect for more than three-quarters of a century. Contemporary physical theory differs profoundly from classic Newtonian physics on the important matter of how the consciousness of human agents enters into the causal dynamics of empirical phenomena. The new quantum principles contradict the older idea that mechanical processes alone can account for all observed empirical data. Contemporary quantum physical theory brings directly and irreducibly into the overall causal structure certain psychologically described choices made by human agents about how they will act. This key development is applicable to neuroscience, and it provides neuroscientists and psychologists with an alternative conceptual framework for describing neural processes. The new framework, and specifically the well described physical principle known as quantum Zeno effect, enable scientists and clinicians to better understand the neuroplastic mechanisms relevant to the growing number of studies demonstrating the capacity of directed attention and mental effort to systematically alter brain function. Clinical and neuropsychological findings from research on mindful awareness and placebo effect will be discussed and elucidated in light on this theoretical paradigm shift.

SUPERIOR NEUROPROTECTIVE EFFECTS OF NEUROTROPHIC FACTORS IN HEAT STROKE FOLLOWING NANOPARTICLES TREATMENT. A COMPARATIVE STUDY WITH OTHER NEUROPROTECTIVE AGENTS IN THE RAT*

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Abstract: The possibility that a mixture of several neurotrophic factors and active peptides induces superior neuroprotection in nanoparticles induced exacerbation of brain damage in heat stroke was examined in our rat model in compari-son with levertiracetam (44 mg/kg), pregabalin (200 mg/kg), topiramat (40 mg/kg, i.p.) and valproate (400 mg/kg) in rats.

Rats subjected to 4 h heat stress in a biological oxygen demand incubator at 38°C (Rel Humid 45-47 %; Wind vel 22.4 to 25.6 cm/sec) developed behavioral and neuropathological symptoms of heat stroke e.g., profuse salivation, prostration, hypotension as well as breakdown of the blood-brain barrier (BBB) permeability to proteins, brain edema formation and neuronal, glial and myelin damage. These symptoms and brain pa-thology were exacerbated in rats pretreated with engineered nanoparticles from met-als, viz. Cu or Ag (50-60 nm particle size 50 mg/kg, i.p. /day for 7 days before).

Pretreatment with neurotrophic factors daily for 3 days before heat stress in saline treated animals significantly thwarted brain pathology and reduced behavioral symptoms as compared to all other drug treatments in identical manner. However, in nanoparticles treated animals cerebrolysin 5 ml/kg was needed to achieve similar neuroprotection following heat stroke. Interestingly, all the other compounds even in higher doses were ineffective in these nanoparticles treated rats after heat exposure.

These observations are the first to show that neurotrophic factors have the most superior neu-roprotective effects in heat stroke in both normal and nanoparticles treated rats as compared to other contemporary neuroprotective agents, not reported earlier.
*This work was supported by CNCSIS ˆUEFISCSU, project number PNII ˆ IDEI 787/2007, Romania

AMYLOID AND ALZHEIMER'S DISEASE

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Alzheimer's disease (AD) is the most prevalent neurodegenerative disorder globally. AD begins as mild short-term memory deficits and culminates in total loss of cognitive and executive functions. Currently, the precise cause of the disease is not known and there is no cure. Genetic studies have identified mutations in amyloid precursor protein (APP) and presenilin 1 and 2 (PS1, PS2) that cause rare, dominantly inherited familial AD. Proteolytic processing of APP by ß-site APP cleaving enzyme followed by PS-containing y-secretase complex generates amyloid-ß (Aß) peptides that deposit in amyloid plaques. Genetic and cell biological studies show increased production of more amyloidogenic Aß peptides associated with familial AD-linked mutations, provides strong support for the amyloid hypothesis, which posits that Aß peptides play a pivotal role in AD pathogenesis. Indeed, Aß peptides, both extracellular and intracellular are neurotoxic in vitro, and produce AD-like symptoms when administered locally to the brain. However, Aß peptides are also generated as part of normal metabolism. Despite the genetic and biological evidence that supports the amyloid hypothesis, there is a growing body of evidence which shows that Aß peptides are unlikely to be the sole factor in AD etiology. For example, recent neuroimaging studies confirm autopsy findings that amyloid deposits are present in cognitively normal individuals, whereas some AD patients show no amyloid deposits in positron emission tomography scans. It is possible that amyloid-independent mechanisms cause defective endo-lysosomal trafficking, altered intracellular signaling cascades, or impaired neurotransmitter release and contribute to synaptic dysfunction and/or neurodegeneration, leading to dementia in AD. Moreover, tau/tangle pathology, another hallmark of AD, is largely unaffected Aß-lowering strategies, which also suggests that Aß plaque clearance may not halt or slow the progression of dementia in individuals with mild-to-moderate AD. Effective disease-modifying treatments for AD will likely need to evolve from a strategy that addresses both amyloid-dependent and amyloid-independent mechanisms.

PREVENTION OF AGE-RELATED CHANGES IN HIPPOCAMPAL LEVELS OF 5-METHYLCYTIDINE BY CALORIC RESTRICTION

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The molecular basis underlying cognitive decline in normal aging and age-related disorders like Alzheimer's disease is not yet fully understood. Recent studies have suggested that epigenetic mechanisms of DNA methylation may play a pivotal role in mediating alterations of gene expression and associated cognitive function. The hippocampus is a crucial region for learning and memory known to be affected by age-related alterations such as DNA damage and oxidative stress. However, it is so far unknown whether the aging process affects DNA methylation processes in the mouse hippocampus and /or whether caloric restriction or overexpression of antioxidants have an impact on DNA methylation. Aberrant DNA methylation patterns have been linked to molecular and cellular alterations in the aging brain. Caloric restriction (CR) and upregulation of antioxidants have been proposed as interventions to prevent or delay age-related brain pathology. We have shown in large cohorts of aging mice, that age-related increases in DNA methyltransferase 3a (Dnmt3a) immunoreactivity in the mouse hippocampus were attenuated by CR, but not by overexpression of superoxide dismutase 1 (SOD1). Our aims are to investigate age-related changes in immunoreactivity (IR) of 5-methylcytidine (5-mC) and DNA methyltransferase (DNMT) 3a in the mouse hippocampus and the additional effects of caloric restriction and overexpression of the antioxidant Cu/Zn superoxide dismutase (SOD1).

Results

Examination of 5-mC-IR in 12- and 24-month-old wild-type (WT) mice on control diet, mice overexpressing SOD1 on control diet, WT mice on CR, and SOD1 mice on CR, indicated an age-related increase in 5-mC-IR in the hippocampal dentate gyrus, CA3 and CA1-2 regions, which was prevented by CR but not by SOD1 overexpression. Moreover, positive correlations between 5-mC and Dnmt3a-IR were observed in the CA3 and CA1- Further, we observed densely stained DNMT3a cells throughout the whole hippocampus. Stereological quantification revealed an age-related loss in the number of these cells, while caloric restriction attenuated this effect.

Discussion

These findings suggest a crucial role for DNA methylation in hippocampal aging and suggest that the beneficial effects of CR may be mediated, at least in part, via alterations in DNA methylation. This study on the mouse hippocampus shows region-specific age-related alterations in DNA methylation, likely associated with differential DNMT3a-IR. We speculate that the observed alterations have a significant impact on the selective vulnerability of the hippocampal subregions during aging, and by that have functional consequences.

NEUROTROPHIC FACTORS AND SYNAPTIC VESICLE PROTEINS: A COMMON PATHOPHYSIOLOGY IN DEPRESSION AND DEMENTIA?

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Advanced neuropsychiatric research elucidates ever more details of the molecular and cellular pathomechanisms underlying both depression and dementia. Two key protein families involved in the pathological processes associated with these devastating disorders are neurotrophic factors and synaptic vesicle proteins. Interestingly, there seem to be at least partially common pathways, thus possibly explaining to a certain extent the clinical phenomenon that depression is often associated with cognitive deficits, while considerable mood changes are often observed in patients suffering from dementia. This insight may have fundamental consequences for innovative treatment strategies, especially regarding the development and use of compounds which impact on neural plasticity.

INCREASED NEURONAL VULNERABILITY IN NORMAL BRAIN AGEING.

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Current demographic trends show that the significant increase in the proportion of the aged population is set to continue, with an almost doubling in the percentage of those over 65 and a 3-fold increase in the actual numbers of individuals over 85, in the next 3 decades. This outcome is a natural consequence of the advancements in medical science that increased the average life-span of individuals, at least in the developed world.

Ageing is an inevitable part of normal biological development, and it is characterised by a relative decrease in the function and a development of dysfunctions in a variety of organs, including the brain. Ageing is also considered to be a major risk factor for a variety of neurodegenerative disease, such as Alzheimer's or Parkinson's disease and act as an important negative prognostic factor for pathological events such as stroke. An important reason for such co-morbid associations is the increased vulnerability of the aged neurones, such that the pathological processes specific to each disease have much larger effects. Understanding the basis of this increased susceptibility of neurones to injury and death is crucially important in any attempt to reduce the impact of neurodegenerative diseases.

Using an animal experimental model, and both ex-vivo (brain slices) and primary neuronal cultures, we have looked, at the effect of ageing on neuronal vulnerability to a variety of excitotoxic agents – from glutamatergic stimulation, to oxidative stress and mitochondrial inhibition, and show an age-dependent increase in susceptibility to injury for all agents tested. At least a part of this effect is due to mitochondrial dysfunctions, and inhibition of the mitochondrial permeability transition pore reduces some of these effects. In addition, this higher vulnerability is use-dependent in becoming manifest only at relatively high levels of stimulation, while in resting and near- resting conditions there are no significant differences between young and aged preparations.

More recent experiments have shown that the normal ageing involve certain regional heterogeneities, with some hippocampal regions being more sensitive to excitotoxic effects than others. There regional differences might well reflect heterogeneous changes in the distribution and composition of NMDA receptors, with consequent alterations in the types of synaptic plasticity, with a shift in the LTP-inducing mechanism from a NMDA-dependent to a Ca2+-dependent process. In addition, studies from invertebrate animal models suggest that the heterogeneity of the ageing phenotype can be identified to the level of single, individual neurones, mediated by cell-specific changes in gene expression, controlled in an epigenetic fashion.

NOVEL THERAPEUTIC STRATEGIES OF NEURODEGENERATIVE DISORDERS: THE KYNURENINES

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There is evidence that neurodegeneration in Parkinson's disease, Alzheimer's disease (AD) and several other neurodegenerative disorders is mediated, at least partly, by neurotoxic products of the kynurenine (KYN) pathway. Rational therapeutic approaches could be to reduce the expression of these neurotoxic agents or to increase the production of the neuroprotectant kynurenic acid (KYNAC) or make use its analogues. Further research is needed to elucidate the exact role of the KYN pathway in the pathomechanism of these neurodegenerative processes in an effort to promote the development of novel therapeutic agents.

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MODERATE AND SEVERE TRAUMATIC BRAIN INJURY: A PROSPECTIVE MULTICENTRE COHORT STUDY

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Background

Traumatic brain injury (TBI) patterns change constantly as are the approaches in patient management. To evaluate the results of these changes prospective registries can provide valuable information about epidemiologic profiles, injury severity classification and patient outcomes. We studied the epidemiologic profile of patients in a prospective multicentre study. In addition, we examined variation in outcome by comparing findings with earlier multicenter studies and by investigating the effect of injury severity stratification methods. In many observational studies and treatment trials 6 months is usually a study endpoint. We assessed outcome at both 6 and 12 months after injury to address the question whether long term changes in outcome occur beyond 6 months post-injury.

Methods

In a multicentre study (POCON) we collected detailed data on demography, aetiology, injury severity variables, initial CT findings, management and outcome of patients with moderate and severe TBI in the Netherlands.

Results

Five level 1 trauma centers included 508 consecutively admitted patients (≥16 years, 69.5% male, mean age 47.3 years). A total of 339 (66.7%) were diagnosed with severe and 169 (33.3%) with moderate TBI based on the admission Glasgow Coma Scale (GCS) score, though in at least 15% this classification would have been different if the injury scene GCS was chosen for injury severity stratification. Road traffic accidents constituted the main injury cause (50.6%) and initial intracranial CT abnormalities were detected in 72.9% of all patients. Admission to an intensive care unit occurred in 82.6% with severe and 43.2% with moderate TBI, an ICP monitor was inserted in 37.5% with severe and 8.3% with moderate TBI and craniotomy was performed in 23.6% with severe and 10.7% with moderate TBI. At 12 months post-injury, 41.6% of the patients with severe and 17.2% of the patients with moderate TBI had died. In almost half (46.2%) of the surviving patients outcome changed between 6 and 12 months post-injury.

Discussion

Compared to earlier studies, a higher mean age and a higher rate of patients without initial CT abnormalities was found in our cohort. Mortality rates in our study remained high emphasizing a continuing need for research of modifiers of patient outcome and of potential therapeutic interventions.

UNRESPONSIVE WAKEFULNESS SYNDROME (UWS)- PROPOSAL FOR A NEW TERMINOLOGY OF APALLIC SYNDROME / VEGATIVE STATE

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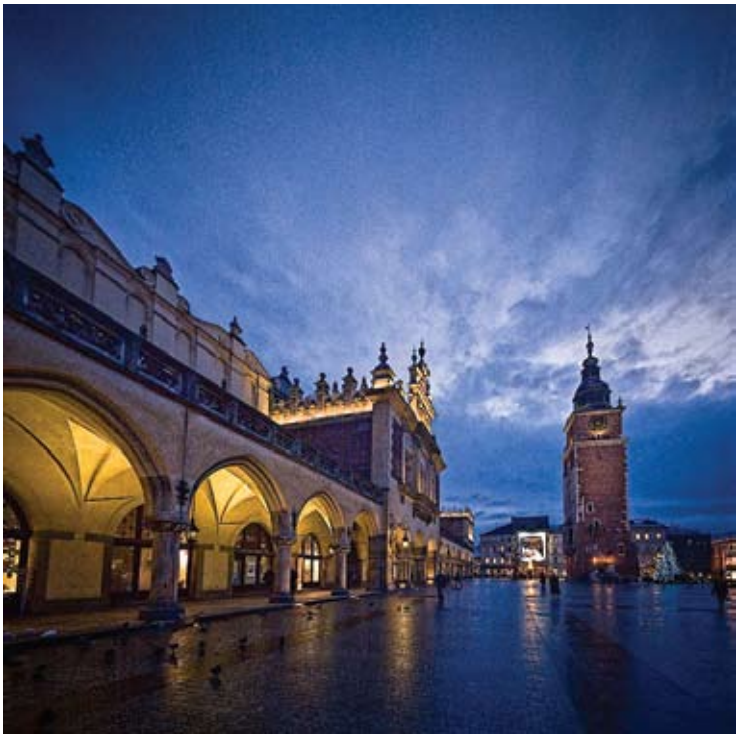
Apallic Syndrome (AS) is a term still used in Central and East Europe and Asia, Vegetative Sate (VS) in the UK, US and in the English literature.
Patients and Method: Full state AS/VS could be present either as a remission defect or an irreversible end stage. For Europe the prevalence of AS/ VS in hospital cases is 0.5–2/100.000 population/year, one-third caused by traumatic brain damage, 70% due mainly to intracranial haemorrhages, tumours, cerebral hypoxemia following cardiac arrest, and chronic neurological diseases.
Results: Full stage AS / VS is clinically defined in three domains (a) anatomy, (b) behaviour, and (c) consciousness (self-awareness). Apallic syndrome describes patients who are awake but unresponsive secondary to severe brain damage. Apallic cannot be explained by or taken for a conditio sine qua non of an anatomically completed and permanent disconnection of neocortical structures and higher cerebral functioning "Vegetative" State must not be persistent or permanent when "vegetative " was chosen to refer to the preserved vegetative (autonomous) nervous system. Our International Task Force on the Vegetative State proposed to recommend Unresponsive Wakefulness Syndrome (UWS as the new nomenclature for the pathological behavioural syndrome as to enable scientists and clinicians to assess all stages accurately regarding neurophysiology, neuropathology, quality management, prognosis, and ethics.
Conclusions: Unresponsive Wakefulness Syndrome (UWS) should replace AS/VS nomenclature since it describes best this specific syndrome.



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Dr. Alvarez has 22 years experience in Basic and Clinical Research on Alzheimer's disease. He was involved in more than 150 research projects, including projects funded by Public Institutions, pharmaceutical R&D studies, industrial and R+D+I projects, epidemiological studies and two projects funded by the European Comunity: (1) MimoVax: Alzheimer's disease treatment targeting truncated AB40/42 by active immunisation (an STREP -Specific Targeted Research Projects- Project approved through the Six Framework Programme of the European Community to develop and test a vaccine for Alzheimer's disease). Period: 2006-2010. (2) BIOMED-PL-950523-European Concerted Action on Pick's Disease. Period: 1995-1998.

As a result of the research activity developed during this period, Dr. Alvarez published more than 120 scientific articles in national and international journals and books. In addition, Dr. Alvarez is actively involved in several scientific forums of his specialty (Congresses, Research Groups, Scientific Journals and Associations).

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PATENTS:
"Multimodality Instrument for Tissue Characterization" US Patent #6,718,196 (April 6, 2004) to NASA on behalf of RW Mah and RJ Andrews.
"Carbon Nanotube-based Nanoelectrode Array for Deep Brain Stimulation" Patent Application by NASA on behalf of J Li, M Meyyappan, R Andrews, NASA Ames Research Center, March, 2003.

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600 Congresses in Greece and abroad, in 380 as invited speaker
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24 books in Neurology, Neuropathology, Neuropsychology, Neurology and the Arts, History of Neurology

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Member of American Neurological Association
President of the International Society for the amelioration of the quality of life in chronic neurological diseases
President of the Medical Association for support of health and medical education in Asia and Africa.

DISTINCTIONS

11 Gold medals

TITLE

Archon Actuarius of the Ecoumenical Patriarchate of Constantinopolis.

OVIDIU
BĂJENARU
/ROMANIA

1983 : M.D. at the Faculty of Medecine of University of Medecine and Pharmacy "Carol Davila" Bucharest
1989 : specialist in neurology, confirmed by the Ministery of Health of Romania
1993 : Ph.D. at the University of Medecine and Pharmacy "Carol Davila" Bucharest
1999 (since) : Professor of Neurology at the University of Medicine and Pharmacy " Carol Davila" Bucharest, Chairman and Head of the Neurology Department of the University Hospital of Emergency Bucharest
2000-2004 : Vice-Dean of the Faculty of Medecine - University of Medecine and Pharmacy "Carol Davila" Bucharest
2001(since) : President of the Romanian Society of Neurology
2003 – 2009 : member of the Scientific Committee of ECTRIMS
2004 - 2009 : Member of the Executive Committee of the European Society of Neurology
2008 (since) : Romania official delegate in UEMS – EBN (Board of Neurology)
*sept. 2010 : elected Sectretary of the Executive Committee of UEMS-EBN

POST GRADUATE TRAINING:
1992 - 1994 : post graduate training in clinical neurology and functional investigations of the nervous system at University "Rene Descartes"(Paris)

FIELDS OF INTEREST FOR THE SCIENTIFIC RESEARCH
- stroke, dementia and neurodegenerative diseases (in particular Alzheimer and Parkinson's disease), multiple sclerosis
- more than 300 scientific papers published and reported in different national and international scientific meetings, 5 medical books and monographies (published in Romania), co-author (1 chapter) to the "International Neurology - A Clinical Approach", Wiley-Blackwell, 2009; Principal Investigator in 12 research grants from the Romanian National Council for Science and Research, Coun try Principal Investigator in an International Program of Research for genetic factors in stroke patients; Country Principal Investigator – in more than 30 international, multicentric clinical trials; Principal Investigator of the research site – in more than 30 international and national multicentic trials

AFFILIATION:
- Romanian Society of Neurology (president), European Neurological Society European Stroke Organization, European Federation of Neurological Societies, American Academy of Neurology, Romanian Brain Council (foundation member), Danube Neurological Association (member in the Board), New York Academy of Sciences, American Academy for Advancement in Science, Movement Disorders Society

HEINRICH BINDER /AUSTRIA

EDUCATION:

1953 - 1957	Primary School „Piaristen“ in Vienna, Austria
1957 - 1965	Secondary School Bundesgymnasium VIII, Vienna
1965 - 1972	Faculty of Medicine at the University Vienna MD since (promotion on) 1972, June 6th
1972 - 1978	University Hospital for Neurology, graduated in Medical Specialist for Neurology and Psychiatry
9/1982	Docent for neurology, a title corresponding to PhD
since 1988	Professor for Neurology, University Vienna founding member of the Austrian Society for Neurorehabilitation
5/1989	Head of the Neurological Hospital “Maria Theresien-Schlössel”)
1994-2007	Head of Ludwig Boltzmann Insitute for Restorative Neurology and Neuromodulation
Since 2008	Deputy Head of Landsteiner Institute for Neurorehabilitation and Space Medicine
since 2002	

Head of the Neurological Center, Otto Wagner Hospital, Vienna.
Main focus: Patients with severe neurological/neuropsychological deficits and invasive neurorehabilitation methodscurrently
President of Austrian Society for Neurorehabilitation
President of European Federation Neurorehabilitation Societies
Member of the Management Committee of WFNR

Main topic of research: Neurorehabilitation, brain injury, spinal cord injury, vegetative state/ apallic syndrome (more than 140 publications)

NATAN
BORNSTEIN
/ISRAEL

EDUCATION

1970-73	University of Sienna, Medicine, Sienna, Italy
1973-79	Technion Medical School, Hifa, Medicine, MD, 1979

Date of receiving specialixation certificate: 11 September, 1984
Title of Doctoral dissertation: Dextran 40 in acute ischemic stroke
Name of Supervisor: Dr. Jacob Vardi

FURTHER EDUCATION

1978-83	Tel-Aviv University, Sackler Faculty of Medicine, neurology (residence), Israeli Board certified in Neurology, 1983
1979-83	Tel-Aviv University, Sackler Faculty of Medicine, Post graduate studies in Neurology
1984-87	Sunnybrook Medical Center, University of Toronto, M.R.C stroke, Fellowship

ACADEMIC AND PROFESSIONAL EXPERIENCE

1982-1995	Tel-Aviv University, Neurology, instructor
1991-present	European stroke Conference (ESC), Executive committee
1995-1999	Tel-Aviv University, Neurology, Senior lecturer
1995	Eliprodil CVD 715 clinical trial, Steering Committee
1995-1997	International Stroke Study (IST), Steering Committee
1995-1999	American Academy of Neurology, Member of the International Affairs Committee
1996	Asymptomatic Carotid Stenosis and Risk of Stroke(ACSRS), Advisory Committee
1996-present	The Mediterranean Stroke Society (MSS), President
1996-2002	EFNS, Management Committee
1997-2009	Israeli Neurological Association, Secretary
1999-present	Tel-Aviv University, Neurology, Associated Professor
2001- present	European Society Neurosonology and Cerebral Hemodynamics (ESNCH) Executive committee
2005-present	Neurosonolgy Research Group, Executive committee
2006-present	European Master in Stroke Medicine, Member of faculty
2006-2008	NEST II clinical Trial, Steering Committee
2006-present	SENTIS clinical Trial, Steering Committee
2006-present	CASTA Trial, Steering Committee
2006-present	Brainsgate clinical Trial, Steering Committee
2008- present	World Stroke Association (WSO), Vice president
2009-present	Israeli Neurological Association, Chairman
2009-present	European Stroke Organization (ESO), Member on the board of directors
2010-	NEST III clinical Trial, Steering Committee

PROFESSIONAL ACHIEVEMENTS- EDITORIAL BOARD

1991-present	Neurological Research Journal, Guest Editor
1991-present	STROKE, Member of the editorial board
1998-present	European Journal of Neurology, Member of the editorial board
1999-present	Journal of Cerebrovascular disease, Member of the editorial board
2000-present	Journal of Annals of Medical Science, Consulting Editor
2001-present	Journal of Neurological Science (Turkish), Member of the editorial board
2001-present	Acta Clinica Croatica, Member of the editorial Counsil
2003-present	Italian Heart Journal, International Scientific Board
2003-present	Journal of Neurological Sciences, Guest Editor
2004-present	Turkish Journal of Neurology, International Advisory Board
2005-present	Archives of Medical Sciences (AMS) , Member of the Editorial Board
2006-present	Journal of Cardiovascular Medicine, International Scientific Board
2006-present	International Journal of Stroke, Editorial Board
2006-present	Acta Neurologica Scandinavica, Editorial Board
2009-present	American Journal of Neuroprotection& Neurogeneration (AJNN) Member of the Editorial Board
2010	Neurosonology, International Editorial Board
2010	Frontiers in Stroke, Review Editor

PROFESSIONAL ACHIEVEMENTS- REVIEWER

1998-present	Lancet, Ad Hoc reviewer
1998-present	Diabetes and its complications, Ad Hoc reviewer
1999-present	Journal of Neuroimaging, Reviewer
1999-present	Journal of Neurology, Ad Hoc reviewer
2000-present	Neurology, Ad Hoc reviewer
2003-present	Israeli Medical Association Journal (IMAJ), Reviewer
2003-present	Acta Neurologica Scandinavica, Ad Hoc reviewer
2006-present	Journal of Neurology, Neurosurgery & Psychiatry, Reviewer
2010-	European Neurology, Ad Hoc reviewer

MEMBERSHIP IN PROFESSIONAL SOCIETIES

1977-present	Israeli Medical Association
1983-present	The Israeli Neurological Association
1985-present	Stroke Council of the American Heart Association (Fellow)
1986-present	American Academy of Neurology
1986-present	Neurosonology Research Group of the World Federation of Neurology
1987-present	Stroke Research Group of the World Federation of Neurology
1990-2008	International Stroke Society
1995-2008	European Stroke Council
1995-present	Mediterranean Stroke Society (MSS)
1998-present	European Neurosonology Society
2005-present	World Stroke Organization (WSO)
2008-present	Fellow of the European Stroke organization (FESO)

ANCA
BUZOIANU
/ROMANIA

OCCUPATION OR POSITION HELD
-Professor in Pharmacology
-Dean of the Medical Faculty, University of Medicine and Pharmacy "Iuliu Hatieganu" , Cluj-Napoca, Romania
-General Secretary of the Romanian Society for Pharmacology. Therapeutics and Clinical Toxicology

TITLE OF QUALIFICATION AWARDED
-Specialist in Pediatrics
-Specialist in Clinical Pharmacology
-Senior Clinical Pharmacologist
-PhD

AWARDS IN THE LAST 2 YEARS
-Great "Iuliu Haţieganu" Award of the University of Medicine and Pharmacy Cluj-Napoca 2007
-"Victor Papilian " University Award for fundamental sciences 2006

MAIN ACTIVITIES AND RESPONSIBILITIES
-Head of the Department of Pharmacology (medical, scientific, and administrative responsibilities)
-Chairman of University Department (teaching courses for undergraduate students, postgraduate students and PhD students)
-Dean of the Medical Faculty of the University of Medicine and Pharmacy "Iuliu Hatieganu" Cluj-Napoca (administrative tasks, universitary management, curriculum planning etc)

International journal publications cited in databases
-4 articles
Articles published in Romanian journals, cited in international databases
-6 articles
Papers published in Romanian journals
-46 articles
Monographies
-2 monographies
Chapters in published books
-9 chapters

ORGANISATIONAL SKILLS AND COMPETENCES
-European Society of Clinical Neuropharmacology- member in International Advisory Board
-The Society for the Study of Neuroprotection and Neuroplasticity (SSNN)
-Member European Association of Clinical Pharmacology and Therapeutics
-Member in the Balkan Medical Union
-Member in the International Association for the Study of Pain
-Vice-president of the Romanian Ministry Commission of Clinical Pharmacology, Toxicology and toxic dependences
-Member in The Romanian Group for Therapeutic Guidelines Elaboration
-Executive general secretary of the Romanian Society for Pharmacology, Therapeutics and Clinical Toxicology
-Member in the Ethical Committee of the "Iuliu Hatieganu" University Cluj-Napoca
-Head of the Pharmacology Department
-President of the Deans Romanian Association of Medical Faculties

RUDOLPH J. CASTELLANI
/USA

Post Graduate Education and Training
1990-1993 Residency in Anatomic Pathology, Wayne State University, Detroit, Michigan
1993-1995 Neuropathology Fellow, Case Western Reserve University, Cleveland, Ohio

Certifications
1997 American Board of Pathology, Anatomic Pathology and Neuropathology

Employment History
1995-1999 Assistant Professor, Pathology, University of Maryland School of Medicine
1996-1999 Neuropathology Consultant, Baltimore VA Medical Center
1997-1999 Head of Neuropathology, University of Maryland Medical System
1999-2002 Assistant Professor, Case Western Reserve University, Cleveland, Ohio
2002-2005 Associate Professor, Michigan State University, Department of Human Pathology,
East Lansing, Michigan
2003-2005 Faculty member, Neuroscience Program, Michigan State University
2003-2005 Faculty member, Comparative Medicine and Integrative Biology Program, Michigan State University
2005-present Professor, Pathology, University of Maryland School of Medicine
Rudolph J. Castellani, M.D.

Professional Society Memberships
1993-present College of American Pathologists
1996-present American Association of Neuropathologists
2002-present Neurotoxicity Society
2007-present Society for Neuroscience

Honors and Awards
1995 1995 “Best Attending Pathologist”, University of Maryland Division of Anatomic Pathology. Singular annual award given out by pathology house officers.
1997 1997 “Best Attending Pathologist”, University of Maryland Division of Anatomic Pathology. Singular annual award given out by pathology house officers.
1998 “Wall of Fame,” University of Maryland, Baltimore School of Medicine. One of only
several awards among all sophomore teaching faculty, given by sophomore medical students.
1999 “Wall of Fame,” University of Maryland, Baltimore School of Medicine. One of only
several awards among all sophomore teaching faculty, given by sophomore medical students.
2005 Educator of the Year, Departments of Neurology and Ophthalmology, Michigan State
University. Singular annual award among faculty of the Department of Neurology and Ophthalmology, by vote of house officers.
2007 Teaching Commendation, Pathophysiology and Therapeutics II, University of Maryland
School of Medicine Class of 2010. Award given by Pathology Department based on medical student evaluations.
2009 Golden Chair Award, American Association of Neuropathologists, Best Management of
a Scientific Session, San Antonio, Texas, June, 2009

CORNEL CĂTOI /ROMANIA

Occupational field and career - Veterinary Medicine - Pathology
1992 – 1995 – Teaching assistant, 1995 – 1998 – Assistant, 1998 - 2006 – Associate professor, from 2006 Professor, Department of Pathology, Faculty of Veterinary Medicine, University of Agricultural Sciences and Veterinary Medicine (USAMV) Cluj-Napoca, Romania;
Since 2002, Doctor in Veterinary Medicine; Title of PhD thesis « Study of spontaneous and induced atherosclerosis in animals ». PhD coordinator.

Research field of interest – Veterinary and comparative pathology (atherosclerosis, obesity, liver fibrosis, oncology, Helicobacter associated gastritis); until now, coordinator and member in over 15 Romanian and international research projects.

Academic position – Vice Rector of USAMV Cluj-Napoca.

Membership in national scientific societies - Romanian Cell Biology; Romanian Society of Comparative Oncology; Romanian Association of Wild Animals Pathology; Center for Study of Biocompatibility of Natural and Synthesis Products; Romanian Society of Comparative and Experimental Pathology – president;

Membership in international scientific societies - European Society of Veterinary Pathology.

Publication - books
1. CĂTOI C.- Veterinary Diagnostic Necropsy, Academicpres Ed., Cluj-Napoca, 2003;
2. A.I. BABA, C. CATOI – General Morphopathologie, Academicpres Ed., Cluj-Napoca, 2003;
3. CĂTOI C. – Special Pathological Anatomy - vol. 1, Academicpres Ed. Cluj-Napoca, 2006.
4. BABA AI, C. CATOI – Comparative Oncology, Romanian Academy Ed., 2007, ISBN: 973-27-1457-3 – Online offered by National Center for Biotechnology Information (NCBI)

Scientific papers - 5 articles published in ISI impact factor Journals; 52 articles ISI Web of Knowledge

Manager of 3 Project financed by European Social Fund in Romania.

A.V. CIUREA
/ROMANIA

Honorary President of the Romanian Society of Neurosurgery, Chairman Neurosurgical Clinic 1, "Bagdasar-Arseni" Hospital, 10, Av. Berceni, Bucharest 4, ROMANIA

EDUCATION

1958-1964	- Medical School (IMPh - Institute of Medicine and Pharmacy) of Bucharest,
1972	- Specialist in neurosurgery, Neurosurgical Clinic, Hospital "Dr. Gh. Marinescu",
1976	- "Doctor in Medical Sciences" (Ph.D.), Neurosurgical speciality
1979	- Senior Neurosurgeon, M.D., in Neurosurgical Clinic Hospital "Dr. Gh. Marinesco"
1989	- Chairman of 1st Neurosurgical Department, Neurosurgical Clinic, "Prof. Dr. D. Bagdasar" Hospital, Bucharest.
1993	- Associate Professor UMPh (University of Medicine and Pharmacy), "Carol Davila" Bucureşti, Neurosurgical Clinic 1, "Prof. Dr. D. Bagdasar" Hospital, Bucharest.
1997-2005:	- Director of "Bagdasar-Arseni" Clinical Hospital, Bucharest
1997-present	- Professor of Neurosurgery UMPh (University of Medicine and Pharmacy), "Carol Davila" Bucureşti, Head & Chairman Neurosurgical Clinic 1, Hosp. "Prof. Dr. D. Bagdasar - Arseni" Hospital, Bucharest.

PROFESSIONAL AFFILIATIONS

1980	- Member I.S.P.N. (International Society of Pediatric Neurosurgery)
1982	- Member E.S.P.N. (European Society of Pediatric Neurosurgery)
1994	- New York, New York Academy of Sciencies. Member I.D. 383884 - 4.
1995	- Member International Society for Pediatric Skull Base.
1997	- Munster, EMN (Euroacademia Multidiciplinaria Neurotraumatologica)
1998	- Marsilia, Member SNCLF (French Society of Neurosurgery).
1999	Neurorehabilitation Committee of the WFNS Memmber CNS (Congress of Neurological Surgeons) President of the Romanian Society of Neurosurgery (RSN)
2001	General Secretary of the EMN (Euroacademia Multidisciplinaria Neurotraumatologica) International Honorary Member of Executive Committee of ACNS (Asian Congress of Neurological Surgeons)
2002	CNS (Congress of Neurological Surgeons) Ambassador.
2003	- Secretary, Founder Member AMN (Academia Multidisciplinaria Neurotraumatologica)
2005	- Vice-President WFNS "at large" (World Federation of Neurosurgical Societies). - President, Society of Neuro-Oncology from Romania (SNOR).
2007	- Corespondent Member of Romanian Scientists Academy
2008	- Vice-President AMN (Academia Multidisciplinaria Neurotraumatologica) - Doctor Honoris Causa Medical University "N. Testimitianu", Chisinau, Republic of Moldavia - Honorary President of the Romanian Society of Neurosurgery (RSN)
2009	- Member of Romanian Academy of Medical Science - Vicepresident of EMN (Euroacademia Multidiciplinaria Neurotraumatologica)
2010	- Chairman of WFNS Nominating Committee

László Csiba was born in 1952, Sajószentpéter, Hungary. Now he is the Chairman of Department of Neurology of University Debrecen and Chair of Board of Director's (European Stroke Organisation), President of European Society of Neurosonology and Cerebral Hemodynamics. He is the chair of European Cooperation Committee of EFNS.

His research interests are stroke and stroke-prone diseases, ultrasonic studies in cerebrovascular diseases and clinicopathological studies on cerebrovascular diseases. He published numerous papers on stroke and stroke-related diseases, associated editor of Frontiers on Stroke and member of editorial committee (Intern. J Stroke)

LÁSZLÓ CSIBA
/HUNGARY

ANNA CZŁONKOWSKA /POLAND

Prof. Anna Członkowska MD, PhD finished Medical Academy in Warsaw in 1966. She has been working in the Institute of Psychiatry and Neurology in Warsaw from 1969, and since 1985, she is the Head of the 2nd Department of Neurology. She carried out a lot of fellowships, for example in the Guy's Hospital in London or Max Planck Institute of Psychiatry in Munich and Martinsried.

Constantly she is cooperating with many neurological centers all over the world. Her main interests are: stroke (epidemiology, treatment, rehabilitation), neuroimmunology (mutiple sclerosis, local and systemic immunity in neurodegeneration), Wilson's disease. She was coordinating the National Program for Prevention and Treatment of Stroke and the neurological part of the National Cardiovascular Disease Prevention and Treatment Program in years 1997-2008.

Professor Anna Członkowska is the member of Polish Academy of Arts and Sciences, member correspondent of Polish Academy of Science of American Academy of Neurology, American Neurological Association, the German Society of Neurology and she is Fellow of the Royal College of Physicians of Edinburgh. In 1996-99, she was the President of the Polish Society of Neurology.

She was promoting 32 doctor theses, from her team 4 persons successfully finished habilitations.

Professor Anna Członkowska is an author/co-author of over 250 original papers, mainly published in international journals, as well as author/co-author or co-editor of many books.

VIDA DEMARIN /CROATIA

Professor Vida Demarin, MD, Ph.D. graduated from School of Medicine, University of Zagreb, Croatia, where she gained her Master of Science thesis and Doctor of Philosophy degree. She finished her residency in neuropsychiatry in Sestre milosrdnice University Hospital, Zagreb, Croatia.

In 1994, she was elected Head of Department of Clinical Neurology and Center for Neurological Sciences and Brain, and has run successfully the Department ever since. Under her leadership the Department became Reference Center for Neurovascular Disorders and for Headaches of Ministry of Health of Republic of Croatia.

She is a full member of Croatian Academy of Sciences and Arts. She published more than 600 papers in national and international journals, organized and participated in numerous symposia, seminars, conferences and congresses. She mentored numerous Doctor of Philosophy and Master of Science theses, research fellows, residents and students. She is an investigator in national and international programs, and neuropharmacological trials.

Professor Demarin's field of interest is studying vascular disorders and cerebral autoregulation in neurology. She is a pioneer of neurosonology in Croatia, and is active in the field of stroke prevention. She is the founder of Summer Stroke School – Healthy Lifestyle and Prevention of Stroke that has been organized in Dubrovnik, Croatia since 1990.

She was founder and the first president of Croatian Society for Neurovascular Disorders and Croatian Stroke Society, whose workgroup published national recommendations for stroke management in 2001 and in 2006. She initiated national program of stroke management, organization of stroke unit network and thrombolysis therapy. She is the national coordinator of SITS (Safe Implementation of Thrombolysis in Stroke) trial.

For several decades she leads and organizes national stroke and cerebrovascular disease prevention programs, educates citizens, general practitioners and neurologists. All her projects are devoted to raising health awareness and improving the quality of life. She is herself oriented and promotes healthy living habits, exercise, and Mediterranean diet, setting an example to the public. She authored numerous publications dedicated to lifestyle improvement, disease prevention, and issued a Mediterranean style cookbook with explanations and suggestions on healthy eating.

Professor Vida Demarin is member of numerous Croatian and international professional societies, secretary general of Kuratorium of International Neuropsychiatric Pula Congresses, and president of Central and Eastern European Stroke Society and the Secretary of the WFN Organization and Delivery of Neurological Services Research Group. She is a President of Croatian committee of European Association for Medicine and Art.
She is member of the Executive Board of the Academy of Medical Sciences of Croatia; Fellow of American Academy of Neurology, Fellow of American Heart Association and Fellow of European Stroke Organization She is a member of World Stroke Organization, International Headache Society and more.

MOHAMED S. EL-TAMAWY
/EGYPT

This is to Certify that Dr. Mohamed Soliman Mohamed El-Tamawy born on 5/1/1952 has been graduated from this faculty and obtained the following qualifications:

M.B., B Ch. in November 1975 was signed up very good .
MSc. of Neurology and Psychological Medicine in November 1980 and was signed up Very Good .
MD. of Neurology in May 1984 .

He has been also appointed the following posts :-
House Officer Cairo University Hospitals from 1/3/1976 to 29/2/1977 .
Resident in Cairo University Hospitals from 1/3/1977 to 29/8/1980 .
Clinical Demonstrator in Department of Neurology, Faculty of Medicine, Cairo University from 30/8/1980-5/6/1981.
Assistant Lecturer in Department of Neurology, Faculty of Medicine, Cairo University from 6/6/1981 - 30/10/1984 .
Lecturer in Department of Neurology, Faculty of Medicine, Cairo University from 31/10/1984-28/11/1989.
Assistant Professor in Department of Neurology, Faculty of Medicine, Cairo University from 29/11/1989 - 29/11 /1994 .
Professor in Department of Neurology, Faculty of Medicine, Cairo University from 30/11/1994 - 16/9/2009 .
Chairman of the Department of Neurology, Faculty of Medicine, Cairo University from 17/9/2009 till now .

ANWAR ETRIBI
/EGYPT

Basic Qualification
1969 M.B.Ch.B, Faculty of Medicine Ain Shams University, Cairo, Egypt

Higher Qualifications
1972 D.P.M.&N. Ain Shams University
1978 Medical Doctor (Neurology), Ain Shams University
1979 M.Sc., Psychopharmacology, University of Manchester, U.K.
1982 PhD., Manchaster

Posts
H. President: Egyptian Society of Neurology Psychiatry & Neurosurgery
Past Chairman: Department of Neurology Ain Shams University
Founder&Chairman: Brain Care Company
Director: Heliopolis Neurocenter
Honary Chairman: Pan Arab Society (PAUNS)

EXUPERIO DÍEZ TEJEDOR
/SPAIN

EDUCATION

1974/Jun Universidad de Valencia	MD	Medicine
1977/Nov Universidad Autónoma de Madrid	Neurologist	Neurology
1987/Nov Universidad Autónoma de Madrid	PhD	PhD

PROFESSIONAL EXPERIENCE

- CLINICAL:

Department of Neurology. Hospital Universitario La Paz. Madrid. Resident Doctor 1975
Training in stroke field: Stroke Unit. Department of CNS. Prof. V. Hachinski. LHSC-WOU. London Ontario. 1992
Attending Doctor 1979
Divission Head. 1989-2004
Stroke Unit Director from 1995
Head of Neurology Departmentfrom 2005

UNIVERSITY: Medicine Department (Neurology). Universidad Autónoma de Madrid.
1979 Assistant Professor
1987 Associated Professor
2008 Professor

SCIENTIFIC SOCIETIES. MEMBERSHIP
- SOCIEDAD ESPAÑOLA DE NEUROLOGÍA. From 1978
  - Coordinator CVD study group 1995-1997
  - INTERNATIONAL STROKE ASSOCIATION: Active Member: September 1992.
- EUROPEAN STROKE COUNCIL
- Academic and clinical Member: March 1994.
- Scientific Committee: from 2000
- FESC (Fellow of The European Stroke Council): from 2005
- STROKE COUNCIL OF AMERICAN HEART ASSOCIATION.
  - Active Member from May 1999
  - International fellowship of the American Heart Association (FAHA): from 2001
- EUROPEAN NEUROLOGICAL SOCIETY. Active Member: Septiembre 1990.
- EUROPEAN FEDERATION OF HEADACHE SOCIETY. Active Member: 1994
- INTERNATIONAL HEADACHE SOCIETY. Full Member: Sep. 1996
- AMERICAN ACADEMY OF NEUROLOGY: Corresponding Member. April 1998
- AMERICAN NEUROLOGICAL ASSOCIATION:Active member: 2007
- SOCIEDAD IBEROAMERICANA DE ENFERMEDAD CEREBROVASCULAR
  - Founding member. Sept 1998
  - Vicepresident. Oct 1999- Oct 2001
  - President: Oct 2001- Oct 2003
  - Permanent council: from Oct 2003

INTERNATIONAL JOURNALS. EDITORIAL BOARDS
- CEREBROVASCULAR DISEASES
  - Editorial Board Member: 1997-2005
- STROKE
  - Referee from 1997
  - Associate editor, Stroke en Español. From 2002

- INTERNATIONAL JOURNAL OF STROKE
  - Editorial Board member 2006

HONORS
- "Rafael Hervada" Award of Biomedical Research. Edition 1996. IMQ San Rafael. 27 Feb 1997.
- Award of Clinical Research. Pfizer Fundation. 5ª Edition. E Díez Tejedor, J Tejada y cols. Trabajo: "Does a relationship exist between carotid stenosis and lacunar infarction?". Nov. 2004.
- Nacional Award of Research "Ciudad de Zamora-Excmo. Ayuntamiento" Stroke. Clinical and Basic Research.. Zamora, 16 Nov 2006.

ANTONIO
FEDERICO
/ITALY

Married, two children

Prof. Antonio Federico, born in Polla (Sa) on the 25.08.48, from 1990 is full professor of Neurology at the University of Siena , Director of the Unit Neurometabolic Disease and of the Research Center for diagnosis, therapy and prevention of Neurohandicap and Rare Neurological Diseases.

He was Director of the Department of Neurological and Behavioural Sciences, University of Siena (2002-2008).

He received the degree in Medicine and specialization in Nervous and Mental Diseases, summa cum laude, at the University of Naples in 1972 and 1975.
His biological training was in the Centre de Neurochimie of CNRS, in Strasbourg, directed by prof. Mandel where he worked in the years 1973-75. He after collaborated with many international research groups, in different countries where he spent in the past years some times: in Montreal (Prof. Andermann, Karpati and Shoudgbridge), in London (dr A. Harding and prof. Morgan-Hughes), in Toronto (dr.Robinson), in Bonn (prof. von Bergmann) , in Paris (dr.Baumann), in Baltimore (proff. Moser and Naidu), in Oxford (prof. Matthews), etc.
His clinical formation was made at the Medical School of the University of Naples, in the Dept, Neurology, and after in Siena, where he moved on 1980. Associated professor in Neurology in 1982, since 1990 he is full professor of Neurology, Medical School, University of Siena.

His present positions are:
- full professor of Neurology, University of Siena, Medical School
- Director of the Section Neurological Diseases of the Department of Neurological and Behavioural Sciences of the University of Siena,
- Chairman of the Panel of Neurometabolic Diseases of the European Federation of Neurological Societies, where he was also member of the Scientific Committee.
- Italian Delegate to the World Federation of Neurology, and member of the by-low and Constitution Committee and of the Nominating Committee of WFN
- President of the Italian Society of Neurology
- He is associated editor of the Neurological Sciences, Springer-Verlag, Editor-in chief of the Italian Edition of Continuum ; he is also in the editorial board of several other italian and foreign neurological journals
- From 1995 he is Director of a PhD Programme on Applied Neurological Sciences at University of Siena, from 2004 of the European PhD Programme and European School of Doctorate of Applied Neurological Sciences.
- He is Italian member of European Union of Medical Specialists, in the section Neurology.

In the years 1990-96 he was Secretary of the Italian Society of Neurology.

He coordinated the Study Group on Clinical Neurogenetics of the Italian Society of Neurology.

He has been referee for projects evaluation in the area of Orphan drugs and Orphan diseases for Biomed Projects from EU, for MURST, CNR and Istituto Superiore di Sanità, and other national and international funding agencies, etc.

He is member of the Second Opinion Group of the American Leucodistrophy Association.

He is author of more than 500 article (more than 300 of them quoted by Pubmed). He is author of a chapter on Cerebrotendinous Xanthomatosis, Vinken and Bruyn Edts, Handbook of Clincal Neurology, vol 49, Neurodystrophies and Neurolipidoses. On the book McKusick's Mendelian Inheritance in Man,. Ed.1992, Catalog of Autosomal Dominant and Recessive Phenotypes he is cited for 3 different diseases. He was editor of the book Late Onset Neurometabolic diseases (A.Federico, K. Suzuki and N.Baumann Edts), Karger 1991, and many others book from Italian and international Publishing Companies.

His main field of interest is related to neurometabolic, neurodegenerative and rare diseases, investigated from a genetic, metabolic, neuroimaging and clinical point of vue.

ŞTEFAN FLORIAN
/ROMANIA

Education & Positions Held
• June-2001: Ph.D. degree of the Faculty of Medicine, University of Medicine and Pharmacy "Iuliu Hatieganu" Cluj-Napoca, : "The neurosurgical treatment of the pituitary adenomas"
• 2002 -present: Head of the Department of Neurosurgery, Cluj County Emergency Hospital
• 2001 – 2005: Director of the Cluj County Public Health Institute
• 2003 – 2010: Vice-president of the Romanian Society of Neurosurgery
• 2006- present: Vice-dean of the Faculty of Medicine
• 2007-present: Full professor of the Faculty of Medicine, University of Medicine and Pharmacy "Iuliu Hatieganu" Cluj-Napoca,
• October 2010: President of the Romanian Society of Neurosurgery

Scientific & research activity
Scientific activity: 38 article published in extenso in the country, 70 papers as abstracts, 12 articles published abroad and 28 papers published as abstracts; furthermore 53 posters presented in the country and abroad at different scientific events (course, conferences, and congresses). Author and co-author of 13 books and treaties dealing with neurosurgery topics (treatment options in pituitary adenomas, in intracerebral hemorrhage)

Research line in neurosurgery-oncology (the evaluation of the therapeutical effect of Temozolomide and antiangiogenetic factors in glioblastoma multiforme; the evaluation of the therapeutical effect of the Calpaine inhibitors and angiogenetic factors in spine cord injuries) can be enumerated.

Rich organizing activity aiming the continous medical education of neurosurgerons, from which the annual CME Course for resident doctors since 2003, two neuro-oncology conferences (National Conference of Neurooncology with International Participation in 2007, 2008), the annual German Romanian Neurosurgery Courses (2009,2010, the forthcoming course in May 2011), promoter and organizer of the 1st Regional Congress of the Danube-Carpathian Region in the period of 24th -27th May 2011, at Cluj-Napoca, Romania.

TOMASZ
GASZYŃSKI
/POLAND

1992-1998 – studies at Medical Faculty of Medical University In Lodz.

1999-2000 – postgraduate studies at Humanistic-Economic University in Lodz the Faculty of Administration in Health Service .

1999 – assistant in Department of Anesthesiology and Intensive Therapy, Barlicki University Hospital in Lodz

1999-2002 – postgraduate studies for candidates for doctor`s degree at Medical University of Lodz

2002 – assistant at Chair of Anesthesiology and Intensive Therapy, Medical University in Lodz .

2004 – award of second degree for scientific achievements in 2003 founded by Rector of Medical University of Lodz .

2005 - award of first degree for scientific achievements in 2004 founded by Rector of Medical University of Lodz

2006 – specialist in Anesthesiology and Intensive Therapy.

2006 – assistant professor at Chair of Anesthesiology and Intensive Therapy, Medical University in Lodz .

2006 – graduate of the International School of Instructors in Anesthesia of WFSA

2007 – European Society of Anesthesiology Teachers Grant Award

2008 – associate professor of Anesthesia at Chair of Anesthesiology and Intensive Therapy, Medical University in Lodz

- author and coauthor of over 120 articles in polish and international papers, chapters in manuals for doctors and students.
- a co-founder and president of Airway Management Section of Polish Society of Anesthesiology and Intensive Therapy.

ANDRZEJ
GLABINSKI
/POLAND

Prof. Andrzej Glabinski graduated at Medical Faculty of Silesian Medical University in Katowice, Poland in 1986. In 1987 he moved to Lodz and became an Assistant Professor at Department of Physiology, Medical University of Lodz (MUL), Lodz, Poland. In 1990 he became an Assistant Professor and from 1998 an Associate Professor at Department of Neurology, MUL. In 2005 he organized at MUL Department of Experimental and Clinical Neurology and became its Chairman. In 2007 he was promoted to the position of Professor at MUL. From 2008 he is a Chairman of Department of Propedeutics of Neurology, MUL and a Vice-Dean for Education at Faculty of Biomedical Sciences and Postgraduate Education, MUL.

Prof. Glabinski defended his PhD thesis in 1992 and habilitation in 2000 at the Medical Faculty of MUL. He was a Research Fellow at Department of Neurosciences of the Cleveland Clinic Foundation, Cleveland, OH, USA in the laboratory of prof. R.M. Ransohoff from 1993 through 1996. The major focus of his research are studies on pathogenesis of multiple sclerosis, especially on the involvement of chemokines in this process. He has published more than 40 papers in international journals including J. Autoimmunity, J. Immunol, Am. J. Pathol, Brain Behav. Immunity, Clin Exp Immunol, Scan J. Immunol, etc. He has got many international and national research grants. Additionally to research he is involved in many educational and clinical activities at MUL, Lodz, Poland. He is also practicing neurologist working in university hospitals in Lodz.

VLADIMIR
HACHINSKI
/CANADA

Vladimir Hachinski, CM, MD, FRCPC, DSc, Doctor honoris causa3 is Professor of Neurology and Distinguished University Professor at the University of Western Ontario, London, Canada. He pioneered stroke units (with John W. Norris), and discovered (with Stephen Oppenheimer and David Cechetto) a heart control center in the brain. He developed the most sensitive, specific and widely used method of identifying the vascular component of cognitive impairment in the elderly (The Hachinski ischemic score, over 2200 citations). He leads a research effort to integrate knowledge from epidemiological, clinical and experimental studies with the aim of improving stroke prevention and delaying Alzheimer disease. He was the Editor-in-Chief of the journal STROKE, the leading publication of the field and is President, World Federation of Neurology and member of the Order of Canada. Recently he received the Ontario Premier's Discovery Award in the Life Sciences and Medicine.

HASSAN S. HOSNY /EGYPT

Dr. Hassan Hosny is currently a Professor of Neurology at Cairo University (since 1992). After completing his residency training at Cairo university hospitals , he left to the USA where he completed fellowships in clinical Neurophysiology and epilepsy at the University of Chicago and the Cleveland clinic foundation (1988-1991). Dr Hosny's main interest is Epilepsy as well as movement disorders and headache .

He is the Secretary of the Egyptian epilepsy Association which he founded in 2004.He works closely with the international league against epilepsy (ILAE) where he chaired the commission on East Mediterranean affairs (CEMA) till 2009.He also sits on the board of the Educational and Epilepsy care commissions of the ILAE. Dr Hosny is also a member of the Editorial board of the international journal of epilepsy research. He organized several successful international epilepsy congresses in Sharm ElSheikh in 2003 and in Luxor in 2007 , EEG course in 2009 and an epilepsy meeting in Luxor 2010

VOLKER HÖMBERG /GERMANY

MEDICAL DIRECTOR

St. Mauritius Therapy Hospital Meerbusch

PERSONAL DATA

Born 25 July 1954
Married to Priv.-Doz. Dr. Kristina Müller, paediatric neurologist

MEDICAL CAREER

1973 - 1980	Medical School, Universities of Düsseldorf and Freiburg; Elective in Neurology at Boston City Hospital, Boston, Mass.; National Hospital for Nervous Diseases, London
since 1975	Junior researcher in the Department of Neuropsychology at the C. & O. Vogt Institute for Brain Research, Düsseldorf and the Department of Neurology, Freiburg (Prof. R. Jung)
1980 - 1981	Research fellow in the Department of Neuropsychology (Prof. G. Grünewald) at the C. & O. Vogt Institute for Brain Research, Düsseldorf
since 1981	Clinical training in the Department of Neurology (Prof. H.-J. Freund),
Heinrich-	Heine-University Düsseldorf
since 1985	Senior registrar in the Department of Neurology, Heinrich-Heine-University Düsseldorf
since 1987	Senior investigator for the German Research Council Special Task Force in Neurology at Heinrich-Heine-University (SFB 200 and SFB 194)
1987-2005	Medical director of the Neurological Therapy Center (NTC), Heinrich-Heine-University Düsseldorf
since 1988	Board examiner for Neurology at the local examination board (Ärztekammer Nordrhein)
1989-1997	Vice president of the German Society for Neurological Rehabilitation
1993	Habilitation in Neurology, Heinrich-Heine-University Düsseldorf
since 1995	Board examiner for physical medicine and rehabilitation (Ärztekammer Nordrhein)
1997-2005	Medical director of the Neurological Therapy Center, Cologne
1998-2004	President of the German Society for Neurological Rehabilitation
since 2000	Medical director and head of neurology, St. Mauritius Therapy Hospital, Meerbusch
since 2003	Secretary General World Federation for NeuroRehabilitation (WFNR)
since 10/2004	Vice president of the German Society for Neurological Rehabilitation
since 2005	Panel-Chairman Neurorehabilitation for European Federation Neurological Societies (EFNS)

AMOS D. KORCZYN /ISRAEL

Professor Amos D. Korczyn is the Sieratzki Professor of Neurology at Tel-Aviv University. Professor Korczyn graduated from the Hebrew University – Hadassah Medical School in Jerusalem in 1966 (MD), where he also received an MSc degree in pharmacology (cum laude) in 1966. He trained in neurology at Beilinson Hospital and at the National Hospital for Nervous Diseases, Queen Square, London. He was the Chairman of the Department of Neurology at the Tel-Aviv Medical Center since 1981 until 2002. Professor Korczyn has a particular interest in dementia. He has authored or co-authored over 600 articles in peer-reviewed journals, as well as chapters in books, etc. Professor Korczyn is or has been an Editorial Board member of 15 international journals, and organized several neurological conferences, mainly in the field of dementia, Parkinson's disease and other degenerative brain disorders, as well as CONy – the International Congress on Controversies in Neurology.

BORIS KOTCHOUBEY /GERMANY

EDUCATION:

1959 - 1969: School education
1969 - 1975: 1st Moscow Medical Institute, Moscow USSR: M.D. (general physician)
1980 - 1983: Institute for General and Educational Psychology, Moscow USSR. Diploma in Psychology and Ph.D. in Psychology (Psychophysiology).
May 11 1983: Ph.D. conferred with thesis: Effects of hereditary and environmental factors on individual differences in human orienting response.
June 12 2002: Habilitation thesis (required in Germany to apply for full professorship): The relationship between event-related brain potentials and cognitive processes - A critical re-evaluation

EMPLOYMENTS:

1975 - 1979: General physician, First-Aid-Station, Moscow Region USSR.
1979 - 1980: Technical assistant at the Russian Academy for Educational Sciences, Moscow USSR.
1980 - 1986: Research assistant at the same Institute.
1986 - 1991: Senior research assistant at the Institute of Psychology, USSR (now: Russian) Academy of Sciences, Moscow USSR.
1992 - 1993: A. von Humboldt Research Fellowship, University of Ulm, Germany.
1994: Research assistant at the Medical University of Lübeck, Germany.
Since 1995: at the Institute for Medical Psychology, University of Tübingen, Germany.

RESEARCH ACTIVITIES:

Electrocortical processes and behavior
Cognitive psychology and psychophysiology
Psychophysiology of perception and action
Brain self-regulation
Learning processes in the development and treatment of neurological diseases
Altered states of consciousness; disorders of consciousness

TEACHING:

1986 – 1988: Introduction into Physiological Psychology, 3rd Moscow Pedagogical School
1990 – 1991: Psychophysiology of Stress and Extreme Conditions, Moscow State University, Faculty of Psychology
1997 – today: Medical Psychology for medical students, an obligatory course, University of Tübingen, Faculty of Medicine
1998 – 1999: Neurophysiological Mechanisms of Perception and Action, University of Tübingen
1998 – 2004: Selfregulation of the Brain, University of Tübingen
2001 – 2002: Electrophysiology of Cognitive Processes, a practical course in English, University of Tübingen, Graduate School
2005: "My beloved enemy, the TV": Real and imagined dangerous of electronic media, University of Tübingen, Faculty of Medicine

2005: Can education be helpful in schizophrenia?, University of Tübingen, Faculty of Medicine
2006: Evidence-Based Medicine or Medicine-Based Evidence?, University of Tübingen, Faculty of Medicine
2006: Do you see, what I don't see? Benjamin Lee Whorf's views on color perception, University of Tübingen, Faculty of Medicine
2007 – 2010: Regret and conscience in the case of erroneous decision making in medicine. University of Tübingen, Faculty of Medicine
2007 – 2009: Fundamental Concepts for Understanding Positions in Mind-Body Problem, University of Tübingen

SUPERVISION:

8 Master Theses in Psychology, one of them running (one from the Moscow State University, the other from the University of Tübingen, Faculty of Cognitive Science)
7 MD Theses, one of them running (one from the University of Ulm, the other from the University of Tübingen, Faculty of Medicine)
12 PhD Theses, one of them running (two from the Russian Academy of Sciences; one from the University of Ulm; one from the Universita degli Studi in Padua, Italy; one from the University of Strasbourg, France; and 7 from the University of Tübingen, Faculties of Biology and Cognitive Science).

MEMBERSHIP IN PROFESSIONAL ASSOCIATIONS:

Society for Psychophysiological Research
British Psychophysiological Society
German Society for Psychophysiology and Its Applications
German Association of University Teachers
Neurex

MACIEJ KRAWCZYK /POLAND

Maciej Krawczyk, PT PhD graduated Faculty of Physiotherapy in Józef Piłsudski's Academy of Physical Education in Warsaw in 1991.
Since 1991 he was working at the Department of Neurology in General Hospital of Warsaw Medical University. Since 1999 he has been a chief physiotherapist at the II Department of Neurology in Institute of Psychiatry and Neurology in Warsaw. He has also been working with students of physiotherapy for last 18 years.
He took part in many postgraduate courses and trainings in the field of neurological rehabilitation in Poland, Great Britain, Germany, Switzerland, Austria, Italy and Portugal.
His main interests are: stroke, multiple sclerosis and restorative neurology.
Since 1999 he is a member of council of specialists in Polish Stroke Program.
From 2007 to 2010 he coordinated scientific Grant Founded by Polish Ministry of Science.
He is an author or co-author of original papers and has promoted 35 master theses.
He has given over 50 postgraduate courses about reeducation of motor functions for physiotherapists.

SIMONE LANG
/GERMANY

Professional Career

- 03/1999: Diploma Psychology, University of Tübingen, Germany

Diploma thesis: "Psychophysiology in patients with severe motor deficits"

- 04/1999-03/2003: Research assistant, Institute of Medical Psychology and Behavioral Neurobiology, University of Tuebingen, Germany (Prof. Dr. B. Kotchoubey): PhD in Psychology, Doctoral thesis: "Cognitive processing in vegetative state: development and implementation of psychophysiological test procedures"
- 04/2003-02/2005: Research assistant, University Hospital Lübeck, Department of Neurology (Prof. Dr. R. Verleger), Germany
- 04/2005-06/2008: Assistant professor, Department of Cognitive and Clinical Neuroscience, Central Institute of Mental Health (ZI) Mannheim (Prof. Dr. Herta Flor), Germany;

Group leader of the SFB-project "Learning and plasticity in posttraumatic stress disorder", University of Heidelberg, Germany

- since July 2008: Assistant professor, Department of Clinical Psychology and Psychotherapy (Prof. S. Barnow), University Heidelberg, Germany: Head of the section "Neuroimaging"

Research projects

- Emotional and cognitive awareness in disorders of consciousness
- Emotion regulation in brain tumour patients
- Emotion regulation, empathy and theory of mind in borderline personality disorder
- Experimental and Clinical Neuropsychology

Methods

FMRI, voxel based morphometry (VBM), ECG, EDA, EEG (event-related potentials)

Lectures

- Neuropsychology
- Medical Psychology
- Clinical Psychology

ROBERTO
MATURANA
/CHILE

Chief of Stroke Unit at the Service of Neurology-Neurosurgery Hospital DIPRECA Santiago Chile

Assistant Professor of Neurology Universidad de Santiago de Chile (USACH)

Board Member of the Society of Neurology, Psychiatry and Neurosurgery (SONEPSYN),

Member of the Cerebrovascular Working Group at SONEPSYN

Chief of Neurorehabilitation Unit at MEDS Medical Center, Santiago, Chile

MERLE G. PAULE
/USA

Dr. Paule received his Bachelor of Science degree in Biochemistry and his Ph.D. in Pharmacology and Toxicology at the University of California at Davis after which he conducted post-doctoral studies in Behavioral Pharmacology and Toxicology at the University of Arkansas for Medical Sciences. In 1983 he began work at the FDA's National Center for Toxicological Research in Jefferson, Arkansas, where he remains today. In, 2000 Merle attained certification as one of FDA's Senior Biomedical Research Scientists and in 2005 became the Director of the Division of Neurotoxicology at NCTR. Dr. Paule has played a major role in developing an automated system for monitoring multiple complex brain functions in nonhuman primates, children, and rodents. These functions include learning, short-term memory, motivation, color and position discrimination and time perception and are used as measures for determining the effects of drug and other chemical exposures. Utilization of similar or identical behavioral tasks across species serves to facilitate the interspecies extrapolation of exposure data and, thus, the risk assessment process. Merle has served as an elected officer or appointed committee member in several prestigious scientific societies including Past President of the Behavioral Toxicology Society, the Neurobehavioral Teratology Society and the Neurotoxicology Specialty Section of the Society of Toxicology. He has also served the NBTS as a member of the Publications, Constitution and By-Laws, and Nominations Committees and as its representative on the National Advisory Committee of the 'Decade of Behavior.' Dr. Paule is a member of several other scientific societies including the Society for Neuroscience, the Society of Toxicology and the American Society for Pharmacology and Experimental Therapeutics. He is a reviewer for several scientific journals and sits on the editorial boards of Neurotoxicology, Neurotoxicology and Teratology and the Journal of Toxicology and Environmental Health. Merle has published over 160 research articles and 25 book chapters and holds Adjunct Professorships at the University of Arkansas for Medical Sciences in the Departments of Pharmacology and Toxicology and in Pediatrics. Dr. Paule is an elected Fellow in the Academy of Toxicological Sciences and in the International Behavioral Neuroscience Society.

DAFIN F. MUREŞANU /ROMANIA

CURRENT POSITIONS

Chairman and Professor of Neurology, Department of Neurology, University CFR Hospital, Cluj Napoca, Romania

Vice Dean of the Faculty of Medicine, "Iuliu Hatieganu" University of Medicine and Pharmacy, Cluj-Napoca, Romania

President of the Society for the Study of Neuroprotection and Neuroplasticity

Member of the Romanian Academy of Medical Sciences, Romania

OTHER ACADEMIC DEGREES

2002-2004 MBA, School of Health Care Systems Management, The Danube University, Krems, Austria

1998 Specialization in Leadership, The Arthur Anderson Institute, Illinois, USA

PAPERS PUBLISHED IN INTERNATIONAL JOURNALS (INDEXED IN ISI AND PUBMED)
30 articles

PAPERS PUBLISHED IN OTHER JOURNALS, (INDEXED IN OTHER DATABASES)
44 articles

PAPERS PUBLISHED IN ROMANIAN JOURNALS
46 articles

MONOGRAPHS
7 monographs

CHAPTERS IN PUBLISHED BOOKS
5 chapters

Fluent in: English, Italian

ACADEMIC MEMBERSHIPS
INTERNATIONAL SCIENTIFIC SOCIETIES

World Academy for Multidisciplinary Neurotraumatology (WAMN); Chairman of the Scientific Committee (2008-2010); Secretary (2010-present)

Danube Neurological Society; Executive Management Committee

European Society of Clinical Neuropharmacology; Secretary General

European Federation of Neurological Societies (EFNS); Member of the Neurotrauma Panel

Global College for Neuroprotection and Neuroregeneration (GCNN);

Vice-President, Chairman of the Clinical Committee

The Society for the Study of Neuroprotection and Neuroplasticity (SSNN);
Founder and President

European Neurological Society (ENS)
Society for Neuroscience

European Stroke Organization

New York Academy of Science

EDITORIAL BOARD

Frontiers in Neuroscience; Associate Editor

International Journal of Neuroprotection and Neuroregeneration

The Romanian Journal of Neurology

Romanian Journal of Clinical Anatomy and Embryology

Acta Neurologica Transilvaniae

American Journal of Neuroprotection and Neuroregeneration; Guest editor

Journal of Cellular and Molecular Medicine; Guest editor

Journal of Medicine and Life

AWARDS

2010 University of Medicine and Pharmacy Cluj-Napoca, Faculty of Medicine
"Octavian Fodor" Award for the best scientific activity of the year

2009 Romanian Academy "Gheorghe Marinescu Award" for contribution to
neuroprotection and neuroplasticity

2009 Excellence Award; "Viata Medicala Romaneasca" Medical Journal

2007 Award for the best Medical TV Series Program; Romanian Television Channel 2.

GELU ONOSE
/ROMANIA

DEGREES, QUALIFICATIONS AND PROFESSIONAL POSITIONS:

- Professor at the (State) University of Medicine and Pharmacy (UMF) "Carol Davila", in Bucharest, Romania - the M6 Chair
- MD; - PhD; - Scientific Researcher of I-st Degree; Doctorate Conductor
- Senior Physician of : - Physical & Rehabilitation Medicine (PRM) and
  - Gerontology & Geriatrics (G-G)
- Competences in : - General Ultrasonography
  - Health Services Management
- Chief of the M6 Chair of the "C. Davila" UMF and of the PRM Discipline/ (neural-muscular) Clinic Division of UMF, at the Teaching Emergency Hospital "Bagdasar-Arseni" (TEHBA), in Bucharest
- Research-Development Director of TEHBA
- Selected and invited, based on the field of study, by Thomson Reuters and Times Higher Education, to participate, among highly-specialized scholars worldwide, in the annual invitation-only Academic Reputation Survey - part of the Thomson Reuters Global Institutional Profiles Project - that will support the World University Rankings and thus represent, with the response, thousands of peers - as: students, scholars, and administrators (March, 2010)
- A member of the Board of the Romanian Society of Rehabilitation, Physical Medicine and Balneoclimatology

EDITORIAL, SCIENTIFIC, DIDACTIC, MANAGERIAL - ACTIVITIES, ACHIEVEMENTS AND RECOGNITION:

- Seven (7) published books (see below, the list of works) - the last one in October, 2009 - (one of them : "The Spondyloarthropathies", was distinguished and received, in 2002, the "Iuliu Hatieganu" Award of The Romanian Academy)

- Four new books and a chapter within a textbook in preparation (to be published between 2010 - 2011)

- About 200 scientific works and papers - communicated within many national and international congresses, conferences, symposia, etc., and/or published in peer-reviewed or non peer-reviewed medical journals -, scientific phase reports within research projects and professional interviews/ articles, in mass-media

- 2 Invention Certificates, appointed by the State Office for Inventions and Marks (SOIM) and 2 Invention requests - one already accepted by SOIM and published in the Official Bulletin of Intellectual Property (No. 1/ 2008 - list of works)

- Main awards: the "Iuliu Hatieganu" Award of The Romanian Academy (2002); the Award of the National Authority for Scientific Research, for the RDI project, entitled "ACTUAT"(2006); the Gold Medal at the International Saloon of Inventions, Geneve/ Switzerland, for the RDI project acronymed: "MOD" (2008)

- A member of the Grants & Research Department's Scientific Council, of the UMF "C. Davila"
- A member of the Editorial Board of the (peer-reviewed) journal "Infomedica",
- A member of the Editorial Board of the (Romanian) "Rehabilitation, Physical Medicine and Balneology " Review,
- A member of the Editorial Board (in charge with Neurorehabilitation and Scientific

Research) of "Romanian Neurosurgery"- the Romanian Society of Neurosurgery's review
- President of the :
  - Romanian Society for Neurorehabilitation (RoSNeRa) - affiliated to WFNR and AMN
  - Romanian Society of Pathology, Therapy and Rehabilitation in SCI (RoSCoS)
- affiliated to ISCoS and ESCIF
- A member of the :
  - Romanian Medical Association
  - Romanian Society of Physical Medicine & Rehabilitation
  - Romanian Society of Neurosurgery (RSN) - Head of the Neuro-Rehabilitation Section and respectively, of the Research Department of RSN
  - Romanian Society of Biomaterials,
  - Balkan Medical Union (BMU),
  - International Society of Hydrothermal Technique (SITH - the National Council of the Romanian Section SITH - RS),
  - British Society of Gerontology (BSG)
  - The International Spinal Cord Society (ISCoS)
  - World Academy for Multidisciplinary Neurotraumatology (AMN)
  - a member of the Scientific Committee, afferent to the Prezidium
  - World Federation For Neurorehabiliation (WFNR) - a member of the Management Committee

A (FORMER):

- Department Director within the Romanian Fellowship of Physicians (1997-1999)
- General Manager of the State Sanitary Authority of Bucharest (1998)
- Assistant General Manager of the National Institute of Gerontology and Geriatrics "Ana Aslan" (1998-2001)
- National Representative for Key Action 6 - "The Aging People and their Disabilities" (within the Frame Work Program - FP - 5) at the Scientific Directorate of the European Commission, Brussels, 2000
- General Manager of the National Institute of Rehabilitation, Physical Medicine and Balneoclimatotogy (2000-2001)
- Member in Evaluation Panels for Scientific Grant Proposals (of the National RDI Agency or/and of the Romanian Academy, 1999-2005)

JÓZEF OPARA
/POLAND

Born 1940
1960-1967 medical study at Silesian University of Medicine in Katowice.
Graduated in 1967.
Specialties: neurology 1977
medical rehabilitation 1982
Doctorship: 1983 (MD)

Aggregation (polish habilitation, more than PhD): 1997

Professor in Academy of Physical Education in Katowice: since 1998
Chair of Physiotherapy in Neurology

Full Professor in Physical Culture: 2008
Scholarship: Austria – invited by AUVA - Allgemeine Unfallversicherungsanstalt - 1986
Member of European Panels in EFNS: Neurorehabilitation, Neurotraumatology, Post-Polio,
Chairman of the Organizing Committee of the III Congress of Polish Society of Rehabilitation in Cieszyn – Ustron 1998
Leader of European Panel of Neurorehabilitation in EFNS: 1998-2000
Certificate: tutor in Evidence Based Medicine 2001
Member of Editorial Board in Polish Journal of Physiotherapy since 2001
Co-originator and member of council of WFNR: since 2002
Originator of Polish Society for Neurological Rehabilitation 2003

Regional vice-President of World Federation for NeuroRehabilitation for Central and Eastern Europe since 2009

Special scientific interrest:
- stroke rehabilitation
- spasticity
- rehabilitation in paraplegia (SCI), Spondylotic Cervical Myelopathy (SCM), Post-Polio
- clinimetrics
- Quality of Life measures
- rehabilitation after TBI
- rehabilitation in PD
- rehabilitation in MS

BOGDAN O. POPESCU
/ROMANIA

Academic Education and Appointments

1996	MD, 'Carol Davila' University School of Medicine, Bucharest, Romania
1997- 2002	Resident in Neurology, University Hospital Bucharest
2000 -	Assistant Professor, 'Carol Davila' University School of Medicine
2001	PhD, 'Carol Davila' University School of Medicine - suma cum laudae
2002 -	Neurologist, University Hospital Bucharest
2004	PhD, Karolinska Institute, Stockholm, Sweden
2005 -	Head of Laboratory of Molecular Medicine, 'Victor Babeş' National Institute of Pathology, Bucharest, Romania
2008-	Senior Neurologist, University Hospital Bucharest
2009 -	Lecturer, 'Carol Davila' University School of Medicine
2009 -	Senior Researcher, 'Victor Babeş' National Institute of Pathology, Bucharest, Romania

Awards

1999	Beaufour-Ipsen prize for the best research study in neurology
2000	Young histochemist award - International Society of Histochemistry and Cytochemistry
2004	Diploma of scientific merit – 'Victor Babeş' National Institute of Pathology
2007	Romanian Academy award for medical research
2010	'Science and Art National Foundation Award of Excellence for research in the field of Neuroscience and Neuropathology

Other current activities

Guest editor for Alzheimer's review series at Journal of Cellular and Molecular Medicine
Executive editor of Romanian Journal of Neurology
Secretary General of the Romanian Society of Neurology
Research director of the Society for the Study of Neuroprotection and Neuroplasticity
Director, Victor Babeş' National Institute of Pathology, Bucharest, Romania
Spokesman for University Hospital Bucharest

Selected publications

1. Popescu BO, Toescu EC, Popescu LM, Bajenaru O, Muresanu DF, Schultzberg M, Bogdanovic N. Blood-brain barrier alterations in ageing and dementia. J Neurol Sci, 283:99-106, 2009.
2. Popescu BO. Still debating a cause and diagnostic criteria for Alzheimer's disease. J Cell Mol Med. 2007;11:1225-6.
3. Romanitan MO, Popescu BO, Winblad B, Bajenaru OA, Bogdanovic N. Occludin is overex in Alzheimer's disease and vascular dementia. J Cell Mol Med. 2007;11(3):569-79.
4. Cowburn RF, Popescu BO, Ankarcrona M, Dehvari N, Cedazo-Minguez A. Presenilin-mediated signal transduction. Physiol Behav. 2007;92:93-7.
5. Hansson CA, Popescu BO, Laudon H, Cedazo-Minguez A, Popescu LM, Winblad B, Ankarcrona M. Caspase cleaved presenilin-1 is part of active gamma-secretase complexes. J Neurochem. 2006;97:356-64.
6. Popescu BO, Ankarcrona M. Mechanisms of cell death in Alzheimer's disease: role of presenilins. J Alzheimers Dis. 2004;6:123-8.
7. Popescu BO, Cedazo-Minguez A, Benedikz E, Nishimura T, Winblad B, Ankarcrona M, Cowburn RF. Gamma-secretase activity of preseni-

lin 1 regulates acetylcholine muscarinic receptor-mediated signal transduction. J Biol Chem. 2004;279:6455-64.
8. Cedazo-Mínguez A, Popescu BO, Blanco-Millán JM, Akterin S, Pei JJ, Winblad B, Cowburn RF. Apolipoprotein E and beta-amyloid (1-42) regulation of glycogen synthase kinase-3beta. J Neurochem. 2003;87:1152-64.
9. Popescu BO, Oprica M, Sajin M, Stanciu CL, Bajenaru O, Predescu A, Vidulescu C, Popescu LM. Dantrolene protects neurons against kainic acid induced apoptosis in vitro and in vivo. J Cell Mol Med. 2002;6:555-69.
10. Popescu BO, Cedazo-Minguez A, Popescu LM, Winblad B, Cowburn RF, Ankarcrona M. Caspase cleavage of exon 9 deleted presenilin-1 is an early event in apoptosis induced by calcium ionophore A 23187 in SH-SY5Y neuroblastoma cells. J Neurosci Res. 2001;66:122-34.

PIETER
RIEDERER
/GERMANY

Date of birth: 21st March 1942, Königsberg, Germany
1960-1968: Studies on Technical Chemistry at the TU Vienna, Austria; 1970: Doctor Degree; 1979: Associate Professor (Univ.-Dozent) TU Vienna; 1983: titl. A.o. Univ.-Prof. TU Vienna, Austria; 1986 – current; from 1971 to 1986 I was Head of Clinical Neurochemistry at the thereof Ludwig Boltzmann Institute, Lainz Hospital, Vienna, Austria. Since 1986 Univ.-Prof. (University Würzburg); Head, Clinical Neurochemistry; Clinic and Polyclinic of Psychiatry and Psychotherapy, Univ. Wuerzburg, Germany.
After my retirement on April 1st 2010 I am guest professor at the later Institute and University.

14 international awards and Honorary Memberships in Scientific Societies; Member of the Deutsche Akademie der Naturforscher Leopoldina (2007); Honorary Member of the Hungarian Academy of Sciences (2007); Honorary Dr. degree Int. Univ. Catalunya, Barcelona (2008); Involved in several Current International Joint Projects; Current assistance in several scientific Journals;Board Memberships: President of the (DGBP) German Society of Biological Psychiatry 1994-1998; President of the (ESCNP) European Society for Clinical Neuropharmacology (1995/96); President of the (DPG) German Parkinson Society 2000-2004 (Vice president until 2000); Organizing Chairman World Congr. Biol Psych. 2001, Berlin; President 16th Int. Symp. Parkinson's Disease 2005, Berlin; Honorary Membership of the German Society of Biological Psychiatry (2006); President 39th Int. Danube Symp. Neurol Sciences 2007, Würzburg; President 1st Int. ADHD Congress 2007, Würzburg; Honorary President of the German Society of Parkinson's Disease (2007); Member of the Deutsche Akademie der Naturforscher Leopoldina (2007); Honorary Member of the Hungarian Academy of Sciences (2007); Honorary Dr. degree Int. Univ. Catalunya, Barcelona (2008)
Membership in 15 scientific societies; Organizer of numerous symposia, workshops, congresses; Publications and lectures: about 1.000 scientific papers in the field of neurology and psychiatry, including hand-book articles; 20 books relevant to the field of psychiatry and neurology; Head Brain Bank Center Würzburg and BrainNet EuropeII subgroup

Main scientific interest

My main interests in the past have been research projects in the field of neuropsychiatry, especially introduction of selegiline into the treatment strategies of Parkinson's disease (PD) (1974/5), discovery with my co-workers of amantadines and memantines mechanisms of action (1989, 1991), discovery of the role of iron in PD (1985 – present), discovery of the loss of respiratory chain activity (complex I) in PD (1989), development of the concept of clinical neuroprotection (1983 – present), neurodevelopmental aspects of neuropsychiatric disorders (2001 – present), etiological aspects of neurodegenerative disorders.
I have a close relation to clinical research programs, human biochemistry/physiology, biochemical, molecular and pharmacological aspects of pathogenesis of neurodegenerative disorders including post mortem analysis and access to the BrainNet Europe II-initiative. This is reflected by my list of more than 1.000 publications.

BEATA SÁNIOVÁ
/SLOVAKIA

EDUCATION:
Title MD: Faculty Medicine in Martin, Comenius University in Bratislava, 1973.
Board Certified: Anaesthesia and Intensive Medicine I.gr, and II.grade.
PhD. Degree: 1994 – in Neurology. Thesis: The influence the general anaesthesia on high brain functions.
Assoc. Prof. Degree: 1999 in Neurology. Thesis title: Early and late survival patients after intracranial spontaneous bleeding.
December, 8, 2009 the professor appointment (the University Professor) in Czech Republic (Field of study: Anaesthesiology, Intensive Medicine and Algeziology).

CURRENT POSITION:
The head of Clinic of Anaesthesiology and Intensive Medicine (from 1995).

PROFESIONAL EXPERIENCE:
1990- 1995: Assist. Prof. at the Clinic of Anaesthesiology and Intensive Medicine in Jessenius Faculty of Medicine in Martin.

PROFESIONAL AFFILIATIONS:
European Resuscitation Council
International Brain Research Organisation
European Society of Intensive Care
International Stroke Society
Slovak Medical Society
Slovak Association of Anaesthesiology and Intensive medicine
Slovak Association of Neurology
Slovak Association of Clinical Neurophysiology
Slovak Association of Neurosciences of the Slovak Academy of Sciences
Member of the working group for PhD studies in Neurology
Member of Editorial Board of Central European Journal of Medicine

JEFFREY M. SCHWARTZ
/USA

PRESENT POSITIONS
Research Psychiatrist
UCLA Department of Psychiatry and Biobehavioral Sciences Los Angeles, California
Private Practice - Psychiatry

PAST POSITIONS
Attending Physician, 1985-1992
Member, Pharmacy and Therapeutics Committee, 1989-1992
Instructor in Psychopharmacology for PGY-I, II, III
Psychiatry Residents (Full Year Courses), 1985-1989 Cedars-Sinai Medical Center, Los Angeles, CA

EDUCATION
1978 M.D. Downstate Medical Center, Brooklyn, New York
1973 B. A. (with honors) in Philosophy, University of Rochester, Rochester, New York

PROFESSIONAL TRAINING
7/84—7/85 Marjorie Grey Post Doctoral Research Fellow;

UCSF - Langley Porter Psychiatric Institute and UC Berkeley-Lawrence Berkeley
Laboratory with Drs. Enoch Callaway and Thomas F. Budinger

7/82––6/84 Resident in Psychiatry, UCLA–Cedars-Sinai Medical Center, Los Angeles, California 7/80—7/82 NIMH Postdoctoral Fellow with Dr. Floyd E. Bloom;
Arthur Vining Davis Center for Behavioral Neurobiology, Salk Institute, La Jolla, California
6/79—6/80 Resident in Psychiatry, University of California, San Diego, San Diego, California 6/78—6/79 Intern in Medicine Sepulveda Veterans Administration Hospital Sepulveda
California
1/78—6/78 Research Elective in Pharmaceutical Chemistry with Dr. Neil Castagnoli University of California, San Francisco, San Francisco, California
6/77—1/78 Research Elective in Biological Psychiatry with Dr. Leo Hollister Stanford University Stanford, California

MEMBERSHIP
Society for Neuroscience

LICENSURE & BOARD CERTFICATION
California Medical License No. G40933
Diplomate, American Board of Psychiatry and Neurology, February, 1986.

HARI SHANKER SHARMA
/SWEDEN

EDUCATION
1. Higher Secondary 1969 Bihar School Examination Biology,Phy,Chem. IIndBoard Math
2. Intermediate (Sci) 1970 LS College, Bihar Univ. - do- Pass Muzaffarpur
3. B. Sc. (Hons.) 1973 -do- Zoology (Hons) , 2nd Cl.1st Bot, Chem
4. M. Sc. (Cytology) 1977 Bihar University Zoology, Cytology 1st Cl. 1st Muzaffarpur
5. Dr. Phil. (Sci) 1982 Banaras Hindu University Physiol/Zool Awarded Inst. Med. Sci, Varanasi
6. Dr. Med. Sci. 1999 Uppsala University, Neuroanatomy Awarded Med. Fac. Uppsala
7. Dr. Sci. 2009 Univ Med & Pharmacy, Neurobiology Conferred
(Doctor Honoris Causa) Cluj-Napoca, Romania

POSITIONS
1. Jun. Res Fellow 1977-1978 Minist. Science & Technol. Inst Med Sci Research UP State Govt. BHU
2. Sr. Res Fellow 1978-1981 Univ. Grants Commission - do- Research Govt. of India
3. Post Doct. Fellow 1981-1982 Indian Council Med Res -do- Research Govt. of India
4. Res. Associate 1982-1986 Univ. Grants Commission -do- Leader/Res Govt. of India
5. Res. Officer 1986-1986 Council Sci & Tech, (Eqv. Assist. Prof.) Govt. of India
6. Res. Scientist A 1986- Univ. Grants Commission (Equiv. Assoc. Prof.)
7. Visiting Scientist 1988-1989 Uppsala University Inst. Pathology CNS/Res Uppsala, Univ. Hospital
8. Humboldt Fellow 1989-1991 Alexander von Humboldt Free Univ. Leader CNS/Res Foundation, Bonn. Germany West Berlin
9. Res Scientist 1991- Uppsala University Univ. Hospital/ CNS/Res.Lead. BMC CNS-Injury
10. Prof. Neurobiology 1999- Med Res Counc -do- CNS Research Leadership
10. Docent 2004- Uppsala University Univ. Hospital CNS Res/Neuroanat

AWARD YEAR ORGANIZATION SUBJECT/DETAILS
1. Silver Medal 1974 L.S.College, BU, Muzaffarpur 1st position in B.Sc. Hons. (Zoology), L.S.College
2. Gold Medal 1977 Bihar University, Muzaffarpur 1st posiition, M.Sc, Zoology, Bihar University
3. SIRI Research Award 1986 Ind. Assoc. Biomedical Sci. Best Research Paper
4. Shakuntala Amir Chand 1988 Ind. Counc. Med. Res. Best Work on “BBB” during Research Prize 1983-1988
5. Career Award 1988 Univ Grants Commis Neurobiology/Res
6. Neuronal Plasticity Award 1991 Soc. Brain Dysfunct. Best Res Paper, Sicily on “CNS Thermal Plasticity”
7. Ronnöws Prize 1996 Dept. of Human Anatomy Best Res in Neuroanatomy UU 1994-1996
8. Distinguished Leadership 1998 Am Bio Res Assoc Neuroscience Award
9. Hwassers Prize 1999 Uppsala Medical Association Best Thesis in Medical Faculty (Basic Res)
10. Gold Record 1998 Am Bio Res Assoc Outstanding Achievement in Neuroscience
11. Life Time Achievement 1998 Am Bio Res Assoc Neurobiology Res/Citation Award
12. Outstanding People of the 2000 Int Bio Cent, Cambridge, UK Dedication to Neuroscience
20th Century,
13. Certificate of excellence 2005 US FDA/NCTR, Ar. USA Dedication to Neurosci Drugs of Nat Ctr Toxicol Res Abuse Research

14. DISCA 2006 NIH/NIDA Dedication on Research on Drugs of Distinguished International Scientist Collaboration Award (DISCA) Abuse
15. DISCA (IInd time) 2007 NIH/NIDA -do- National Institute on Drug Abuse
16. Outstanding Researcher Award 2007 EOARD, London, UK Research on Nanoparticles in
Eur Off Aerospace Res Dev Blood-Brain Barrier
17. Hall of Fame, Neuroscience 2009 Am Bio Res Inst Dedication to Neuroscience Research/
Citation/Novelty
18. Neuroscience Icon 2010 DST/New Delhi, India to be awarded

STEPHEN D. SKAPER
/ITALY

CAREER: NIH Postdoctoral Fellow, Department of Medicine, University of California, San Diego (1973-1976); Fellow in Human Genetics, Department of Pediatrics, Case Western Reserve University, Cleveland, Ohio (1977); Postgraduate Research Biologist, Department of Biology, University of California, San Diego (1978); Assistant Research Biologist, Department of Biology, University of California, San Diego (1979-1982); Associate Research Biologist, Department of Biology, University of California, San Diego (1983-1987); Head, Laboratory of Neuropharmacology, Neuroscience Research Laboratories, Fidia S.p.A. - Abano Terme, Italy (1987-1993); Principal Scientist and Head, Laboratory of Cell Biology, Researchlife S.c.p.A. (a Lifegroup Company), Biomedical Research Center, St. Thomas Hospital, Castelfranco Veneto (TV), Italy (1993-1996); Visiting Professor, Department of Pharmacology, University of Padova, Padova, Italy (1997); Assistant Director, Molecular Neurobiology Research, SmithKline Beecham Pharmaceuticals, New Frontiers Science Park, Harlow, United Kingdom (1998-2001); Senior Team Leader, Migraine and Stroke Research, Neurology & GI Centre of Excellence for Drug Discovery, GlaxoSmithKline R & D Limited, Harlow, United Kingdom (2002-2003); Senior Team Leader, Neuro Cell Sciences/Neurodegeneration Research, Neurology & GI Centre of Excellence for Drug Discovery, GlaxoSmithKline R & D Limited, Harlow, United Kingdom (2004-2007); Senior Team Leader, Target Validation Dept (Cognition and Pain), Centre of Excellence for Drug Discovery, GlaxoSmithKline R&D Limited, Harlow, United Kingdom (2008); Adjunct Professor, Department of Pharmacology and Anesthesiology, University of Padova, Faculty of Medicine, Padova, Italy (2009-present).
PROFESSIONAL MEMBERSHIPS: Sigma I (The Scientific Research Society); Phi Lambda Upsilon (honorary chemistry society); Alpha Chi Sigma (professional society in chemistry/chemical engineering); Society for Neuroscience

JOURNALS EDITED: Editor-in-Chief, CNS & Neurological Disorders – Drug Targets; Associate Editor, American Journal of Neuroprotection and Neuroregeneration
REVIEW PANELS: The Wellcome Trust (UK), Medical Research Council (UK), Biotechnology and Biological Sciences Research Council (BBSRC) (UK), Austrian Science Fund (ad hoc review panel to evaluate interdisciplinary doctoral programmes in neuroscience), Dutch Internationale Stichting Alzheimer Onderzoek (The Netherlands), National Science Foundation (US), The Alberta Heritage Foundation (Canada)
RESEARCH INTERESTS: Molecular biology and cellular mechanisms of cell death in CNS aging and neurodegenerative disorders and neuroinflammation.Track record of drug discovery project leadership in kinases, ion channels, G-protein-coupled receptors, DNA repair enzymes, growth factors, identification and optimization of tools for target validation studies, utilising RNAi, conditional and viral knockdown\outs\ins, transcriptomics, proteomics and in vitro cell-based disease or mechanism relevant assays in rodent systems.
PUBLICATIONS: OVER 230 publications in the neurosciences, including book chapters and symposia proceedings.
PATENTS: Pharmaceutical compositions containing monosialoganglioside GM1 or derivative thereof suitable for the treatment of Parkinson's disease (Patent No.: US 6,620,792 B1), use of CRF receptor agonists for the treatment or prophylaxis of diseases, for example neurodegenerative diseases (US 2003/0186867 A1), treatment of conditions with a need of GSK-3 inhibition (PCT WO 02/062387 A1), use of CRF receptor agonists for the treatment or prophylaxis of diseases, for example neurodegenerative diseases (PCT WO 01/72326 A1), use of monosialoganglioside GM1 or N-dichloroacetyl-lyso-GM1 for preventing or reversing neuronal degeneration induced by long term treatment with L-DOPA in the therapy of Parkinson's disease (EP 0 770 389 A1)

HARRY STEINBUSCH /THE NETHERLANDS

EDUCATION

April-1982: Ph.D. degree (Cum Laude) of the Faculty of Medicine of the Catholic University, Nijmegen entitled: "Serotoninergic Neurons in the Central Nervous System of the Rat"
1996 - : Full Professor in Cellular Neuroscience; Director of the European Graduate School of Neuroscience
2003 - 2013 Director School for Mental Health and Neuroscience
1995 - 2015 Director and founder of the European Graduate School of Neuroscience (EURON), a consortium of neuroscience departments within eleven universities in Belgium, Germany and the Netherlands
1992 - Editor-in-Chief and founder of the "Journal of CHEMICAL NEUROANATOMY"
2002 – 2010 Scientific Director of the International Alzheimer Foundation in the Netherlands, Belgium, Germany and France.

Several research lines in biomedical/basic neuroscience can be described. A focus is on the neuroanatomical, pharmacological, physiological and behavioral aspects of development and aging. Our general working hypothesis is that pre/ peri or postnetal stress can lead to depression and this by itself can be an early initiator of neurodegeneration. In addition, neurodegeneration and functional repair are studied in animal models and in human material obtained from patients. Topics are development, plasticity, brain aging and dementia, movement disorders, learning and memory. Research questions have primarily to do with the mechanism of changes in the nervous system in diseases and in development and aging. Participating disciplines are: Animal neuropsychology, genetics, neuroanatomy, neuropathology, neurochemistry, neuroimmunology, animal neuropsychology, molecular cell biology, neurophysiology, developmental neurobiology, neuropharmacology.

Rutten, B.P., Van der Kolk, N.M., Schafer, S., van Zandvoort, M.A., Bayer, T.A., Steinbusch, H. W.M., Schmitz, C. (2005) Age-related loss of synaptophysin immunoreactive presynaptic boutons within the hippocampus of APP751SL, PS1M146L, and APP751SL/PS1M146L transgenic mice. Am J Pathol 167, 161-173. (IF: 6.441)

Temel, Y., Blokland, A., Steinbusch, H.W.M., Visser-Vandewalle, V. (2005) The functional role of the subthalamic nucleus in cognitive and limbic circuits. Prog Neurobiol 76, 393-413. (IF: 11.933)

Kreczmanski, P., Heinsen, H., Mantua, V., Woltersdorf, F., Masson, T., Ulfig, N., Schmidt-Kastner, R., Korr, H., Steinbusch, H.W.M., Hof, P.R., Schmitz, C., 2007. Volume, neuron density and total neuron number in five subcortical regions in schizophrenia. Brain [IF 9.603]. 130, 678-92

Michelsen, K.A., Schmitz, C., Steinbusch, H.W.M., 2007. The dorsal raphe nucleus--from silver stainings to a role in depression. Brain Res Rev [IF 6.236]. 55, 329-42

Brasnjevic, I., Steinbusch, H.W.M., Schmitz, C., Martinez-Martinez, P., 2009. Delivery of peptide and protein drugs over the blood-brain barrier. Prog Neurobiol [IF 9.130]. 87, 212-51

JOHANNES
THOME
/UK

Johannes Thome studied medicine, philosophy and social psychology and obtained his MD/PhD degrees from Saarland University.

After his training as a resident in Psychiatry and Neurology at the University of Wurzburg, he moved to the USA where he became a Postdoctoral Associate at Yale University.

After two years of intensive and highly successful research in the area of molecular neuroscience and psychopharmacology, he returned to his native Germany and worked as Consultant Psychiatrist and Senior Scientist at the Central Institute of Mental Health Mannheim, University of Heidelberg.

From 2004 to 2011, Johannes moved to Wales and settled in Swansea, where he was the Chair of Psychiatry at the University of Wales Swansea.

In March 2011, he has been appointed Chair of Psychiatry and Psychotherapy at the University of Rostock, Germany, where he is also the Director of the Psychiatric University Hospital and its Research Unit.

EMIL C. TOESCU
/UK

Emil Toescu is University Lecturer at Physiology department of University of Birmingham. The current major research direction in the lab is the study of the physiological changes associated with ageing in neurons from the central nervous system. Ageing is a major risk factor for the development of various neurodegenerative diseases, and an understanding of the changes in neurons induced by ageing would provide a better understanding of neurodegeneration. The current research in the lab indicates that normal, physiological ageing of neurons is associated with a significant decrease in the metabolic resistance, centered on a chronic change in the mitochondrial function. Area of inters includes Ageing, Neuronal Signaling, Ca Homeostasis, Mitochondria. Recent 11 publications are axed on those themes.

LÁSZLÓ VÉCSEI
/HUNGARY

Professor and Director of Neurology
1979 MD, Albert Szent-Györgyi Medical University, Szeged, Hungary
1984 Board examination in chemical pathology
1986 CSc (PhD, University of Szeged)
1987 Board examination in clinical neurology
1987-1989 Research fellow in neuroscience, University of Lund, Sweden (PhD, University of Lund)
1989-1990 Research fellow in experimental neurology, Harvard Medical School, Massachusets General Hospital, Boston, USA
1992 DSc, Hungarian Academy of Sciences
1993- Professor and Director of Neurology, University of Szeged
2001-2007 Corresponding Member of Hungarian Academy of Sciences
2007- Ordinary Member of Hungarian Academy of Sciences
2010- Dean of the Medical Faculty, Univ. Szeged
2011- Regional Vice-President of EFNS

László Vécsei graduated with an M.D. from Albert Szent-Györgyi Medical University (currently University of Szeged) and was then awarded his C.Sc. in the behavioural effects of neuropeptides and D.Sc. in the pathogenesis of neurological disorders. He holds a Ph.D. from the University of Lund, Sweden and received a fellowship at the Department of Neurology, Massachusetts General Hospital, Harvard Medical School, Boston, USA. He is Vice-President of the Medical Section of the Hungarian Academy of Sciences, Past-President of the Society of Hungarian Neurologists and of the Hungarian Medical Association, General Secretary of the Danube Symposium for Neurological Sciences and Past-Secretary of the European Society for Clinical Neuropharmacology (ESCNP).He served in EFNS as a member of the Scientific-, Program-, Liasion and European Affairs- and European Cooperation Committees, the 1st and 2nd European Neurology Board Exam Committee, and Editorial Board of European Journal of Neurology. He was President of the Panel of Developmental Neurology and the Educational Committee. He is Chairman of the Local Arrangements Committee of the 15th EFNS Congress (Budapest, 2011). His main interests are: neurodegeneration and neuroprotection, headache, multiple sclerosis and extrapyramidal disorders (especially the role of kynurenines). (Published PUBMED papers: 262; books and monographs: 14; cumulative citation: > 3000 (www.mta.hu), patents: 5).

PIETER E. VOS /THE NETHERLANDS

Pieter Vos is neurologist at the Institute of Neurology at Radboud University Nijmegen Medical Centre, The Netherlands. His research activities are connected with traumatic brain injury, traumatic spinal cord injury and other acute neurological disorders. Focus of the research activities consist of studies aiming to unravel the clinical, biochemical and genetic determinants of neuroplasticity and recovery after mild, moderate and severe traumatic brain injury. Pieter Vos is founder of the Dutch working group on Neurortraumatology. Current international activities are chairman of the scientist panel on neurotraumatology and head of the task force mild traumatic brain injury, both residing under the European Federation of Neurological Societies.

KLAUS VON WILD
/GERMANY

PRESENT APPOINTMENT
Professor (apl) for Neurosurgery Medical Faculty Westphalia Wilhelms- University of Münster,
Professor (apl) for Neurorehabilitation and Re-engineering of Brain and Spinal Cord Lesions,International Neuroscience Institute, INI, Hannover, Institute at Otto-von-Guericke University, Medical Faculty, Magdeburg, Germany
Visiting professor Armed Force and Rheumatic Rehabilitation Hospital EL AGOUZA Military Hospital Centre, Cairo, Egypt; China Rehabilitation Research Centre, CRRC, Beijing, PRCh

MEDICAL EDUCATION - QUALIFICATIONS:

1966	Graduation from the Medical Faculty of the J.W.Goethe-University Frankfurt/ Main
1968	M.D.
1975	Specialist Neurosurgeon, Department of Neurosurgery, Head Prof. Hugo Ruf
1977	Postdoctoral lecture qualification (Habilitation), Dr.med. habil., in Neurosurgery
1977- 1984	Assistant Professor Med. Faculties of the Universities of Frankfurt and Hanover Consultant Neurosurgical Department Academic Public Hospital Nordstadt, Director Prof Madjid Samii, Hanover
1982- 2002	Director Neurosurgical Clinic Clemens Academic Hospital, Med. Faculty Muenster
1984	Professor Medical Faculty University Münster, North Rhine Westphalia, Germany
1993- 2002	Founder & Head Special Department for Early Neurorehabilitation in Neurosurgery, Licence for education and board examination for neurosurgeons of the medical association in Neurosurgical intensive care, Clinical laboratory medicine in neurosurgery, Neuroradiology, Electroencephalography, Treatment of Pain, Physical Training Dr von Wild has personally performed more than 5000 major operations of CNS and PNS lesions with special interest in pituitary adenomas & tumours of the sella region & the cavernous sinus , CPA tumours , tumours of the spinal cord, brain stem cavernomas; Intramedullary tumours of the spinal cord; all kind of spinal surgery. Birth traumatic spinal cord and brachial plexus lesions;transdisciplinary neurotraumatology and functional reconstruction in cooperation with Reconstructive trauma-, Ear-nose and through-, Head and Neck, Thoracic-, Maxilla facial, and Eye surgeons.
At present:	Functional restoration of locomotion in paraplegics by FES implanted neuroprosthesis and via central nervous system- peripheral nervous system (CNS_PNS) by pass grafting procedure following SCI; Neuro modulation of patients in coma and VS State

CLINICAL RESEARCH:
Organizer & President of numerous national and international congresses, workshops and coursesGuidelines On Quality management in neurotraumatology, functional neurorehabilitation, and outcome:

The German Coma Remission Scale (CRS) In Schmidek, HH (ed) 2000: Operative neurosurgical techniques, 4th edition, Vol. 1, Saunders Comp, Philadelphia, US, pp 45-60
Guidelines on Early Neurological-Neurosurgical Rehabilitation

See Acta Neurochir Suppl. 79, 11-19, 2001
Guidelines on Management of Poly -traumatised Patients .
See The German Interdisciplinary Association for Intensive Care Medicine (DIVI) 1998, only in German
Guidelines on Mild Traumatic Brain Injury,
European J. Neurology, 2002, No 9,207-219)
Revised Guidelines on MTBI Early Management,
See EFNS MTBI Taskforce in EFNS Hand book of neurology 2006,
Guidelines on quality management for AS/VS
European Journal of Trauma Emerg Surg. 2007, No3:268-292
The QOLIBRI : Quality of Life after traumatic brain injury assessment tool See von Steinbüchel N et al 2005 in Acta Neurochirurgica Supp.93, pp 43-49

PRESET
Quality management of multidisciplinary neurotraumatology and brain protection
Quality management and amelioration of patients in long-lasting coma and AS/VS
Neuromodulation in paraplegics after SCI; Exernal audit for cell-transplantation
Neuroethics; Long term outcome, HRQoL, and social re-entry following TBI

DISTINCTION
Professor honoris causa (h..c.) for Neurorehabilitation and Reconstructive Neurosurgery Faculty of Physical Rehabilitation at Al Azhar University, Cairo, Egypt
Doctor honoris causa (Dr.h.c.) at the Faculty of Medicine and Pharmacology, „Iuliu Hatiegau“ University, Cluj- Napoca, Romania Honorary (& founding) President EMN, Euroacademy, and AMN, World Academy of Multidisciplinary neurotraumatology;;Honorary President Romanian Society of Neurorehabilitation RoSNeRa ; Corresponding Fellow The Cuban Society of Neurophysiology (SCNFC); Honorary Chairman WFNS Committee Neurorehabil. & Reconstructive Neurosurgery; Honorary Chairman EFNS Panel Neurotraumatology
Honorary Member (former President)German Soc. Neurotraumatology & Neurorehab.
Honorary Member of the Austrian Society , the Lithunian Society, the Polish, the Romanian Society of Neurosurgery, the Russian Federation of Neurosurgical Societies; The Cuban Neurological Society, Egyptian and Pan Arab Societies for Neurorehabilitation, the Japanese Society for Neural Repair and Neurorehabilitation

SCIENTIFIC SOCIETIES / PRESENT DUTIES
Since 2001 WFNR Executive Board , World Federation for Neurorehabilitation,
Since 2009 EBIS 1st Vice-President , European Brain Injury Society,
IANR Scientific Executive Board, International Association of Neurorestoratology
Since 2008/9 Treasurer (Founding Member) International QOLIBRI Society, CNM, International Society for Clinical Neuromusicology, EFNR Europ. Federation Neurorehabilitation
Since 2003 AMN Secretary General, World Academy of Multidisciplinary Neurotraumatology, Director (CEO) kvw neuroscience consulting GmbH Muenster, D
Founding Member & Member of the Presidium: ISRN International Society of Reconstructive Neurosurgery ;
MASCIN, Madjid Samii Congress of International Neurosurgeons; ESCRI Europ. Spinal Cord Research Institute Giorgio Brunelli Foundation, Brescia, Italy;
Founding Member . DANC/GANS German Academy of Neurosurgery; BDNC, German Social Professional organisation of neurological surgeons.

MARCH 31 / APRIL 3 / 2011

KRAKOW | POLAND

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