HELLENIC SOCIETY FOR AMELIORATION
OF THE QUALITY OF LIFE
FOR CHRONIC NEUROLOGIC PATIENTS

9th International Congress on current treatment and therapeutic perspectives in Alzheimer's, Parkinson's disease, MS and Epilepsy

27 - 30 January 2011, Athens - Greece
Hotel King George Palace

Final Program
&
Abstract's Book

Dear Colleagues and Friends

It is with great pleasure and honor that we address this invitation to you to the 9th International Congress for the Amelioration of the Quality of Life in Chronic Neurological Diseases, which is going to be held this year in Athens. Following the success and the high impact of the previous congresses of our Society on the same subject, which were held in Constantinople, Vienna, Alexandria, Odessa, Catania, Marseille, Thessaloniki and Delphi we are now looking forward with much enthusiasm to welcoming you in Athens in order to discuss the new developments in the research, understanding and treatment of the chronic neurological conditions, having also under consideration the very crucial ethical point of view in debilitating and incurable neurological diseases.

This time the Congress is going to be held in Athens, a city which has a unique place in world's history and has played a definite role in philosophy, medicine, poetry, drama, history, mathematics and arts, achieving an amazing perfection in architecture, sculpture and painting.

Solon's first legislation on the equal rights of all the citizens of Athens, no matter their socioeconomic status, laid the foundations of democracy in 594 BC. Cleisthenes in 508 BC increased the power of the demos, by a major reorganization of the political structure, allowing ecclesia (the general assembly of all the citizens) to become the heart of the political life.

In in 490 BC, during the first Persian invasion of Greece under the king Darius I and the leadership of Datis and Artaphernes, Athenians protected Greece and Europe, under the leadership of Miltiades, defeating Persians in Marathon.

Ten years later (in 480 BC) during the second Persian invasion of Greece under the King Xerxes, Athenians defeated the Persian fleet in the decisive naval battle of Salamis under the leadership of Themistocles. As a result king Xerxes retreated to Asia leaving a part of his army under Mardonius, which suffered a dramatic defeat at the battle of Plataea in 479 BC under the leadership of the King of Sparta Pausanias.

Afterwards the Persians made no more attempts to conquer Greece and Europe, been unable therefore to suspend the blossoming of the miraculous Greek civilization and by extension the European civilization per se.

In spite of the Peloponnesian war (431-404 BC), Athens continued to be the undiminished cultural, intellectual and education center in the World, with numerous authorities in philosophy, such as Socrates, his pupil Plato and Aristotle, a scholar in Plato's academy, Epicurus, Zeno, Cleanthes and Chrysippos . Athenian authorities in History such as Xenophon and Thucydides became the models of historiography through the centuries. The tragic poets Aeschylus, Sophocles and Euripides became the universal patterns of dramatic art.

On Athenian democracy, I remember always the statement by Pericles in his epitaph (A speech which was given by him in the winter of 431BC, when Athenians gave a funeral at the public cost to those who had first fallen in the first battles of the Peloponnesian war).

"Our constitution does not copy the laws of neighboring states, we are rather a pattern to others than imitators ourselves. Its administration favors the many instead of the few; this is why it is called a democracy. If we look to the laws, they afford equal justice to all in their private differences; if no social standing, advancement in public life falls to reputation for capacity, class considerations not being allowed to interfere with merit, nor again does poverty bar the way, if a man is able to serve the state, he is not hindered by the obscurity of his condition. The freedom which we enjoy in our government extends also to our ordinary life"(Thucydides, Peloponnesian War Book 2.34-46).

We have therefore the great honour and the real pleasure to invite you in the 9th Congress of the International Society, which is going to be held in Athens from 27th to 30th of January 2011.

The 9th Congress, following the tradition of the previous ones is organized in order to focus attention on chronic neurological diseases and neurodegenerative disorders, while raising strong scientific, ethical and social awareness of these disorders, which deserve special care and understanding. In a parallel way, we would expect the enlargement the communication amongst clinical neurologists and scientific workers in this field.

We wish cordially that the special cultural atmosphere of Athens with the numerous museums and collections of Art from the Cycladic period (3,300BC) to modern art, the special intellectual pursuits integrating the Socratic spirit with the contemporary philosophy and the special historical environment where "every stone is a witness" would leave you a lasting memory.

Stavros J.Baloyannis MD, PhD
Professor and Head of the Department of Neurology
Aristotelian University
Congress President

Board of the International Society for Amelioration of the Quality of Life in Chronic Neurological Diseases

Honorary President: Prof. Dr. Franz Gerstenbrand (Austria)

President: Prof. Dr. Stavros I. Baloyannis (Greece)

Vice President: Prof. Dr. Kurt Jellinger (Austria)

General Secretary: Prof. Dr. Vassiliki G. Costa (Greece)

Special Secretary: Assis. Prof.Dr. Thomas Tegos (Greece)

Treasurer: Assis. Prof. Dr.Euphrosini Koutsouraki (Greece)

Members: Prof. Dr. Pavel Kalvach (Czech Republic)
Prof. Dr. Amos Korczyn (Israel)
Prof. Dr. Laszlo Vecsei (Hungary)

Board of the Hellenic Society for the Amelioration of the Quality of Life for Chronic Neurological Patients

President: Prof. Stavros I. Baloyannis

Vice President: Em. Prof. Dr. Athanasios Arnaoutoglou

General Secretary: Prof. Dr.Vassiliki G. Costa

Treasurer: Assis. Prof. Dr. Euphrosini Koutsouraki

Officers: Em. Prof. Dr. Constantinos Siamoulis
Prof. Dr. Nikolaos Vlaikidis
Assis. Prof.Dr. Thomas Tegos

Organizing Committee

Arnaoutoglou Athanasios
Arnaoutoglou Marianthi
Artemis Nicholaos
Bostanjopoulou Sevasti
Costa Vassiliki
Diacoyannis Ioannis
Fekas Leonidas
Fokas Constantinos
Fotiou Fotios
Giouzepas Ioannis
Hamlatzis Paris
Karlovasitou Anna
Kaprinis Georgios
Koutsouraki Euphorsini
Kyritsis Georgios
Lagos Georgios
Mavromatis Ioannis
Orologas Anastasios
Papadimitriou Alexandros
Papathanasopoulos Pangiotis
Pappa Persephoni
Parlapanis Aemilios
Pelidou Erieta
Piperidou Charitomeni
Plaitakis Andreas
Polyzoidis Constantinos
Siamoulis Constantinos
Sitzoglou Constantinos
Soldatos Constantinos
Taskos Nicholaos
Tegos Thomas
Vassilopoulos Dimitrios
Vlaikidis Nickolaos
Xafenias Demetrios
Zapheiropoulos Aris

International Scientific Committee

Antonarakis S.	Switzerland	Lassmann H.	Austria
Betterman K	U.S.A.	Leszek J.	Poland
Bouras C.	Switzerland	Link H.	Sweden
Cervos-Navaro J	Spain	Maurer K.	Germany
Federico A.	Italy	Mourelatos Z.	U.S.A.
Gardner-Thorpe C	U.K.	Muresanu D.	Romania
Gerstenbrand F.	Austria	Papasosomenos S.	U.S.A.
Giacobini E.	Switzerland	Perry G.	U.S.A.
Gonatas N.	U.S.A.	Portera-Sanchez A.	Spain
Goebel Hans	Germany	Rizzuto N.	Italy
Gustafson L.	Sweden	Rombakis N.	U.S.A.
Hachinski J.	Canada	Sharma H.	Sweden
Hardy J.	U.S.A.	Tabira T.	Japan
Hauw J.J.	France	Tool J.	U.S.A.
Iqbal K.	U.S.A.	Vecsei L.	Hungary
Jellinger K.	Austria	Von Wild K.	Germany
Kalvach P.	Czech Republic	Wisniewski Th.	U.S.A.
Korczyn A.	Israel		

Invited Speakers

Bettermann K.	U.S.A.	Leszek J.	Poland
Bouras C.	Switzerland	Maurer K.	Germany
Federico A.	Italy	Muresanu D.	Romania
Gerstenbrand F.	Austria	Tool J.	U.S.A.
Jellinger K.	Austria	Vecsei L.	Hungary
Kalvach P.	Czech Republic	Von Wild K.	Germany
Korczyn A.	Israel	Wisniewski Th.	U.S.A.

Poster Evaluation Committee

Korczyn A.	Israel
Papadimitriou A.	Greece
Muresanu D.	Romania

Congress Secretariat:

FORUM CONGRESS & TRAVEL
24 Mitropoleos str. 54624 Thessaloniki - Greece
Tel.: +30 2310 257128, +30 2310 243588. Fax. :+30 2310 231849
e-mail: info@forumcongress.com
web page : www.forumcongress.com

THURSDAY 27th of January 2011

18:30-19:30 Satellite Symposium
Current trends and future advances in therapeutic management of Alzheimer's disease

Pathophysiology of Alzheimer's disease: The clinical significance of double inhibition in the management of Alzheimer's disease
P. Ioannidis

Maximizing benefit of acetylcholinesterase inhibitors in Alzheimer's disease
V. Vagenas

19:45 **Opening Ceremony**
Addresses

Opening Lecture: "Marathon, 2500 Years thereafter"
Professor Dr. Dr. Evangelos Chrysos
Secretary General of the Hellenic Parliament Foundation for Parliamentarism and Democracy
Emeritus Professor of Byzantine History, University of Athens
Secretary General of the International Association of Byzantine Studies
corr. Member of the Austrian Academy of Sciences

Recital for traditional musical instruments and Chorus

Welcome Reception

FRIDAY 28th of January 2011

09:00-09:30 Lecture
Chaipersons: K. Jellinger, S. Baloyannis

The offline brain, does it exist? Apallic and locked in syndrome
F. Gerstenbrand, H. Binder, S. Golasszewski, G. Menditti

09:30-10:00 Lecture
Chairpersons: F. Gerstenbrand, D.Vassilopoulos

Heterogenous mechanisms of mild cognitive impairment in Parkinson's disease
K. Jellinger

10:00-10:30 Coffee brake and poster viewing

10:30-13:00 Lectures
Chairpersons: Th. Wisniewski, A. Papademetriou

10:30-10:55 Immunomodulation as a therapeutic a Therapeutic approach for Alzheimer and prion diseases
Th. Wisniewski

10:55-11:20 Evolution of brain pathology in aging
C. Bouras

11:20-11:45 Alzheimer's disease-New approach to pathogenesis and therapy.
J. Leszek

11:45-12:10 The cerebellum in Alzheimer's disease
S. J. Baloyannis

12:10-12:35 Screening the metabolic causes of dementia: bedside Alzheimer's disease

12:35-13:00 Endogenous neuroprotection and neurodegenerative disorders
L. Vecsei

13:00- 15:00 Lunch

15:00 -16:00 Poster Viewing
Chairpersons: V. Costa, J. Lezsek

16:00 -17:30 Lectures
Chairpersons: K. von Wild, P. Papathanasopoulos

16:00-16:20 Molecular genetics and Neurology: an interleation
D. Vassilopoulos

16:20-16:40 Cognitive dysfunction in Multiple Sclerosis.
P. Papathanasopoulos

16:40-17:00 Is there a need to redefine Parkinson's disease
A. Korczyn

17:00-17:20 The limits of evidence based medicine in neurorehabilitation
D. Muresanu

17:20-17:40 Coffee Break

17:40-19:40 Medical Ethics
Round Table Medico-Ethical Aspects concerning Quality of Life
Following Severest Damage of the Central Nervous System (CNS)
Chairperson: F. Gerstenbrand
Speakers: K. Jellinger, K. Von Wild, A. Korczyn, L. Vecsei, S. Baloyannis

21:00-23:00 Gala Dinner

SATURDAY 29th of January 2011

09:00-09:30 Lecture
Chairpersons: F Gerstenbrand, S. Baloyannis

War is Hell
J. Toole

09:30-10:00 **Chairpersons:** J.Toole, L.Versei

10:00-10:20 From the diagnostics to therapy
K. Maurer

10:20-10:40 Social participation of the mentally and physically severe disabled-a contradictoriness of terms
K. von Wild

10:40-11:10 Coffee brake

11:10-13:10 **Chairpersons:** K. Maurer, N. Taskos

11:30-11:50 Retinal Vasoreactivity as a Marker for Cerebral Vessel Disease in Type II Diabetes?
K. Betterman

11:50-12:10 Leukoareosis and its Haemodynamic causes
P. Kalvach ,Peisker T.Keller J,Bartos A

12:10-12:30 Diagnostics in Parkinson's disease: evaluation, clinical correlates and Treatment
S. Bostantjopoulou

12:30-12:50 Nanostructured Li-batteries in Deep Brain stimulation
K. Aifantis

12:50-13:10 Can Magnetic Stimulation Can Modulate Seizures in Epileptic Patients?
Ph. Anninos

13:10-15:00 Lunch

15:00-17:00 Lectures
Chairpersons: K. Betterman, V. Costa
The influence of internal secretion in Multiple Sclerosis
E. S. Koutsouraki

The role of cerebellum in the pathophysiology of dystonia
M. Arnaoutoglou

Indices of carotid plaque instability in asymptomatic individuals based on transcranial Doppler
Th. Tegos

Carotid plaque echostructure: does it play any role in the clinical decision making?
Ath. Giannoukas

Myasthenia gravis: recent developments on its laboratory diagnosis and antigen-specific therapy
S. Tzartos

Athens Association of Alzheimer's Disease and Related Disorders
P. Sakka

Posters

P1.An immunohistochemical study of NMDA receptors in human cerebellum and hippocampus
Euphrosyni S. Koutsouraki, John J. Anastasiades, Vassiliki G. Costa, Stavros J. Baloyannis
Aristotelian University, Thessaloniki, Greece

P2.Levels of Antibodies against Gangliosides GM1, GD1b and GQ1b in Demented Patients
E.Hatzifilippou, E. Koutsouraki, T. Banaki, M.Traka, V.G.Costa, S.J. Baloyannis[2]
Aristotelian University, Thessaloniki, Greece

P3.Broca's area in Alzheimer's disease
Ioannis A. Mavroudis, Luc Flavien Adipepe, Marina Manani, Samouel N. Njau, Vassiliki Costa, Stavros I. Baloyannis
University of Thessaloniki, Greece

P4. Morphological and morphometric alterations of the neurons of Edinger and Westphal nucleus in Alzheimer's disease.
D. F. Fotiou, I. A. Mavroudis, L.F. Adipepe, D. Mytilinaios, K. Tsamis, V. G. Costa, S. J. Baloyannis
Aristotelian University of Thessaloniki, Greece

P5.Fragmentation of nucleoli and morphological changes of the nuclei are related to neuronal loss, dendritic pathology and spinal loss in Alzheimer's disease
I.A. Mavroudis, D.F. Fotiou, M. G. Manani, S. N. Njau, V.G. Costa, S.J. Baloyannis
Aristotelian University of Thessaloniki, Greece

P6.Vascular and blood brain barrier pathology are related to dendritic alterations and spinal loss in Alzheimer's disease.
I.A. Mavroudis, D.F. Fotiou, SN Njau, K. Tsamis, V. Costa, S. J. Baloyannis
Aristotelian University of Thessaloniki, Greece

P7.Human Amygdaloid bodies: Cytoarchitecture and efferent fiber system.
L. F. Adipepe, I.A. Mavroudis, D. F. Fotiou, S.N. Njau, V. Costa, S. J. Baloyannis
Aristotelian University of Thessaloniki, Greece

P8.Cytokine profile in patients with Alzheimer's disease
M. Traka, E. Hatzifilippou, T. Banaki, E. Koutsouraki, V.G. Costa, S.J. Baloyannis.
Aristotelian University, Thessaloniki, Greece.

P9.Cognitive assessment in alcohol-dependent patients and patients with Alzheimer's disease: The distinct neuropsychological profile Theotoka I, Kapaki E, Ilias I, Paraskevas GP, Liappas I.
Athens National University, Athens, Greece.

P10. Functional Status of Brain Hemispheres in Amnestic Mild Cognitive Impairment (aMCI)
Fereshteh Sedaghat[2], Amin Rakhshani, Thomas Tegos, Stavros J Baloyannis
Aristotle University, Thessaloniki and University of Crete, Greece

P11.The Diagnosis Of Cognitive Impairment In Parkinson Disease
I.Theotoka, Athens University,Athens,Greece

P12 Epidemiologic data of Mutliple Sclerosis in Northern Greece during the last thirty years (1979-2008)
E.S. Koutsouraki, A. Fotakidou, M. Arnaoutoglou, T. Gatsios, S. Koukoulidou, V.G. Costa, S.J. Baloyannis
Aristotelian University, Thessaloniki, Greece

P13.Increase of IL-6 levels is related with depressive phenomena in the acute phase of Multiple Sclerosis
E Koutsouraki, E Hatzifilippou , D Michmizos , C Cotsavasiloglou , V Costa , S. Baloyannis
Aristotelian University, Thessaloniki, Greece

P14.Low bone density in women with Multiple Sclerosis
Papakonstantinou S., Sioka X., Pelidou S.-H., Fotopoulos A., Tsouli S., Georgiou A., Kalef-Ezra John, Kyritsis A.
University Hospital of Ioannina,Ioannina,Greece

P15. HLA associations with multiple sclerosis in Greece
I. Kouri, S. Papakonstantinou , V. Mpempes , H. S. Vasiliadis, A. P. Kyritsis, S.-H. Pelidou
University of Ioannina, Ioannina, Greece.

P16.Antibodies against GM1, GD1b and GQ1b in multiple sclerosis patients
E.S. Koutsouraki , E. Hatzifilippou , T. Banaki , V.G. Costa , S.J. Baloyannis
Aristotelian University, Thessaloniki, Greece

P17.Therapeutic rehabilitation of a patient with NMO-positive relapsing longitudinal myelitis: A case report
A. Ploumis, S-E. Pelidou, D. Varvarousis, A. Kyritsis, A. Beris
University of Ioannina, Ioannina Greece

P18.Scientific Research- A Review
Fereshteh Sedaghat[2], Aliasghar Rakhshani, Stavros J. Baloyannis
Aristotelian University, Thessaloniki, Mashhad University of Medical Sciences- Iran, YETOS - Thessaloniki- Greece

P19.Ischemic cerebrovascular accident in the distribution of anterior choroidal artery: a case report
Thomas J. Tegos, Athinodoros Valavanis, Christos Pitsalidis, Alexandros Chatziapostolou, Angeliki Papadimitriou, Stavros I. Baloyannis.
Aristotelian University of Thessaloniki, Greece.

P20.Mixed gliomas with desmoplastic copmponent: An adult case report
Balogiannis J, Syrmos N, Stavrinou P, Magras J, Polyzoidis K.
Aristotelian University,Thessaloniki ,Greece

P21.Treatment of intractable increased intracranial pressure with decompressive craniectomy
Balogiannis J, Syrmos N, Arzoglou V, Paraskevopoulos D, Tsitlakidis A.
1st Neurosurgical Department AUTH, AHEPA University Hospital, Thessaloniki, Macedonia ,Greece

P22. Suicidal behavior in the Psychiatric Department of General Hospital.
Tananaki M, Kiloudis P, Xerras C, Karafyles G, Mavroudis I, Mavridou E, Stathakis I
Papanikalaou General Hospital, Thessaloniki, Greece

P23.Sports and recreation activities for young children in ancient Greece. The young boxers of Akrotiri ,Santorini (Thera)
Nikolaos Ch.Syrmos, Argyrios Mylonas
Aristotle University , Thessaloniki, Greece

P24.Microcephaly in ancient Greece -The Minoan Microcephalus of Zakros
Nikolaos Ch. Syrmos
Aristotle University , Thessaloniki, Greece

P25.Η συνοδεία και το σχέδιο δράσης σαν ένας τρόπος παρέμβασης για την βελτίωση της ποιότητας ζωής των χρόνιων πασχόντων
Δεμίρης Μόσχος, Δ/της Εταιρείας Σπαστικών Βορείου Ελλάδος

P26. Out of sight, out of mind: The role of the hidden object in a novel memory screening test
Sokratis G. Papageorgiou, Department of Neurology, Eginition Hospital
Alexandra Economou, Department of Psychology, University of Athens

P27.Receptive Aphasia as an early symptom of CJD
Makrydakis G., Anyfandi E., Liapis C. Bonakis A., Routsis C., Spilioti E., Papageorgiou SG., Kalfakis N.
First Department of Neurology, Eginition Hospital, National and Kapodistrian University of Athens, Greece.

P28. A case of abortive Hashimoto encephalopathy exhibiting a selective memory deficit.
C. Koros, A. Economou, G. Mastorakos, A. Bonakis, N. Kalfakis, S. G. Papageorgiou
Cognitive Neurology-Extrapyramidal Disorders Unit, 1st University Department of Neurology, Eginition Hospital, Medical School, University of Athens, Greece, Endocrine Unit, Aretaieion Hospital, Medical School, University of Athens, Greece
Department of Psychology, University of Athens, Greece

LECTURES

L1. The offline brain, does it exist ? Apallic and locked in syndrome

F.Gerstenbrand , Vienna, Austria, H.Binder. Vienna, Austria, St.Golaszewski, Salzburg, Austria
G.Menditti, Vienna/Austria
Karl Landsteiner Institute for Neurorehabilitation and Space Neurology,Vienna/Austria

Coma Vigile (Wachkoma), a special form of a coma, is the leading symptom of the Apallic Syndrome/Vegetative State. The patient is aware but without higher and highest brain functions and shows more or less uniform neurological symptoms). Patho-physiologically the Apallic Syndrome can be compared with the physiological brain function of a new born or a young child. An Apallic Syndrome after an acute severe brain damage shows a typical course passing from an initial state to a full state. In a great number of patients a remission is following (Gerstenbrand, 1967). In the remission state the disturbed consciousness in form of a coma vigile shows a reintegration, the motoric deficits and the sensory dysfunctions are rebuilt together with the re-developement of the higher brain functions and the highest brain functions, the cognitive abilities. The Apallic Syndrome after a progressive brain process (Alzheimer Disease etc.) shows a disintegration of all brain functions to the end stage of an Apallic Syndrome (Vegetative State).

The Apallic Syndrome/Vegetative State is not conform with an Off Line Brain. but can be compared with a partly On Line Brain. The irreversible Off Line Brain corresponds to the Brain Death Syndrome. The term Coma is patho-phisiologically not conform with an Off Line Brain because of the various accompanying symptoms, which are not recognized.

Contrary to the Apallic Syndrome/Vegetative the classical Locked In Syndrome shows only a loss of the motoric functions. The patients are aware with sleep/wake rhythm and full active sensory functions. In the extended Locked In Syndrome based on an enlarged lesion to the meso-diencephalic region, sometimes including parts of the thalamus, symptoms of a stupor, parasomnia, hypersomnia, acinetic mutism and thalamic symptoms can be found.

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

L2. Heterogenous Mechanisms of Mild Cognitive Impairment in Parkinson Disease

Kurt A. Jellinger
Institute of Clinical Neurobiology, Vienna, Austria

Cognitive deficits are common in Parkinson disease (PD), but the range of clinical deficits and their structural backgrounds is variable. Mild cognitive impairment (MCI), representing the earliest clinical features of cognitive disorders, according to current criteria include the amnestic and non-amnestic phenotypes (aMCI and naMCI), the latter with multiple-domain and single domain naMCI. These are heterogenous populations, with prodromal Alzheimer disease (AD) and other dementing disorders represented in both groups. Patients with PD have an increased risk to develop MCI and dementia, the frequency of PD-MCI varying between 21 and 62%, single domain being more common than multiple domain impairment. A recent multicenter analysis of 1,346 PD patients revealed an incidence of MCI in 25.8%, affecting various cognitive domains, most frequently memory, visuospatial and attention-executive abilities (1). Neuroimaging methods show hypometabolism in posterior cortical regions, widespread dopaminergic and cholinergic dysfunctions as well as increased cortical amyloid burden (2). The neuropathology of PD-MCI,

according to recent studies (3, 4), is variable, showing brainstem, brainstem-limbic and neocortical Lewy body stages but low Braak AD stages (mean 2.1-2.7), rare neuritic plaques, variable amyloid plaques in cerebral cortex, but no amyloid in basal ganglia, and occasional mild cerebral amyloid angiopathy (CAA). Both CAA and cortical plaques correlates well with multiple-domain MCI, confirming that amyloid pathology may contribute to cognitive impairment in PD. Some cases showed mild lacunar state in basal ganglia. The presently available data on PD-MCI suggest a heterogenous neuropathology, similar to that found in MCI cases without PD, although in these AD is the most common pathology, infarctions, lacunes, and mixed pathologies also occurring in many patients with MCI not related to PD (5). Given the inherent heterogeneity of PD-MCI, further prospective studies in well documented populations using specific biomarkers including amyloid imaging will be an important tool in the diagnosis and prognosis of early cognitive deficits in PD patients including the different subtypes of MCI, and may be a potential for their prevention or effective therapy. This is a key property for future research.

References

- Aarsland D et al. (2010) Neurology 75:1062-69
- Silbert LC et al. (2010) Brain Pathol 20:646-53
- Adler CH et al. (2010) Acta Neuropathol 120:827-8; 831
- Jellinger KA (2010) Acta Neuropathol. 120:829-30
- Markesbery WR (2010) J Alzheimers Dis 19:221-8

L3. Immunomodulation as a Therapeutic Approach for Alzheimer and Prion Diseases

Thomas Wisniewski[a,b,c]
Departments of Neurology[a], Pathology[b], and Psychiatry[c]
New York University School of Medicine

Alzheimer's and prion diseases belong to a category of conformational neurodegenerative disorders. Alzheimer's Disease (AD), the most common cause of dementia globally, is characterized neuropathological by amyloid β (Aβ) deposition as neuritic plaques and congophilic angiopathy, in addition to abnormally phosphorylated tau accumulation as neurofibrillary tangles (NFTs). Today ~35.6 million people have dementia globally with the number expected to rise to 115 million in 2050. Currently most AD treatment approaches in development target only Aβ related pathology with initial results indicating a very minimal clinical effectiveness. Among the therapeutic approaches being developed for AD, “vaccination” shows greatest promise with many on-going clinical trials. However, current vaccination approaches have a number of problems which include: the potential of toxicity from encephalitis (related to excessive cell mediated immunity), the immunological targeting of both the normal and abnormal Aβ, the resistance of vascular amyloid to clearance, as well as tau related pathology not being specifically addressed . We have recently developed a novel immunization approach to treat AD that targets the shared abnormal conformation of both Aβ and tau, in particular the most toxic oligomeric forms, which appears to be highly effective in model animals. Additional approaches we are developing to reduce AD pathology include stimulation of the innate immune system and inhibition of the interaction between apolipoprotein E and Aβ. For prion disease, we have pioneered the use of mucosal immunization to prevent the entry of the infectious agent via the gut. The GI tract is the natural route of infection in many forms of prion disease. We have shown that in animals with a high IgA and IgG anti-PrP response to our vaccine, complete protection from prion infection via an oral route is possible.

We are currently testing this approach for an emerging prion disease, chronic wasting disease, which is epidemic among deer and elk populations in parts of North American and has the potential to spread to human populations. These approaches, which target the abnormal protein conformation that is central to the pathogenesis of AD and prion disease, show great promise as effective, non-toxic therapies.

L4. Evolution of brain pathology in aging

Constantin Bouras (Geneva, Switzerland)

Neurofibrillary tangles (NFT) and amyloid deposits are present in both normal brain aging and Alzheimer's disease (AD). The quantity of hippocampal NFTs is well correlated with age-associated memory impairment, whereas substantial NFT formation in neocortical association areas is the neuropathological hallmark of AD. The distribution and severity of NFT formation shows a strong correlation with the clinical signs. The neuropathological changes associated with normal brain aging and AD affect selective cortical circuits. Moreover, they support the hypothesis that AD symptomatology may be due to the degeneration of a defined set of long projections, eventually progressing to a syndrome of cortical disconnection.
Different types of vascular lesions are also common in the aged brain. To study these lesions and their cognitive effect a vascular score was developed for both microvascular (histological) and small macrovascular lesions (lacunes). The results showed that only some micro- or small vascular lesions as cortical microinfarcts, demyelination and thalamic and basal ganglia lacunes have a cognitive impact. Other lesions as focal cortical and subcortical white matter gliosis or lacunes in the white matter do not seem to influence the cognitive status. Depending on the age of the subjects we observed an important difference: before 85 years the main determinant of vascular cognitive function is represented by thalamic and basal ganglia lacunes and by cortical microinfarcts after the age of 85 years.

L5. Alzheimer's disease - New approach to pathogenesis and therapy

*__Jerzy Leszek__, **Kazimierz Gasiorowski
*University of Medicine , Department of Psychiatry Wroclaw, Poland
**University of Medicine, Department of Basic Medical Sciences, Faculty of Pharmacy

The findings of amyloid-beta(ABeta) deposition in AD brains after death let to the so-called”amyloid hypothesis”. For over a decade, the amyloid hypothesis has so influenced and guided research in the fied of Alzheimer dementia that many workers regard it as the gold standard of scientific investigation. We have extensively reviewed the literature which claims that AD is caused by the deposition of ABeta within structures called senile plaques that invade AD brains and that such plaque formation then leads to further abnormalities within the nerve cells, eventually killing them. We have found little evidence to support this claim and ample evidence to question it. For example, the amyloid hypothesis has been criticized because its research findings up to now have not generated any benefits in the clinical management and treatment of AD patients nor to an understanding of how the elderly are preferentially affected. The three main flaws of the amyloid hypothesis appear to be that: 1)ABeta deposition has not been found to be toxic or cause damage and death of nerve cells in human or animal brain. , 2) the brains of many cognitively normal aged individuals show abundant ABeta containing senile plaques but no clinical signs of Alzheimer's disease, 3) and since there is general agreement that senile plaques containing Abeta are the products of degenerating nneurons, they can not be the cause, since it is axiomatic that a product is the result not the cause of some activity.

By contrast, there is now considerable and still growing evidence from the fields of epidemiology, pharmacology, neuroimaging, clinical medicine, microscopic anatomy, and molecular biology which indicate that non-genetic AD is a vascular disorder whose underlying cause is impaired blood flow to the brain during advanced aging. This evidence can be summarized as follows: 1) numerous epidemiologic studies link AD risk factors such as stroke, heart disease ,hypertension and atherosclerosis to reduced cerebral blood flow, 2) evidence that AD and vascular dementia(VaD) , an acknowledged vascular disorder, share practically all the same risk factors and may benefit from the same treatments, 3) drug therapy reported to improve AD symptoms(including prescriptive drugs now available for AD) all increase blood flow to the brain, 4) people who are likely to develop AD but do not yet show dementia symptoms can be identified by using brain blood flow measurements and brain PET scans, 5) the clinical symptoms are very similar in most AD and VaD patients, 6) parallel abnormalities occur in brain vessels and brain tissue including ABeta laden plaques in AD and VaD patients, 7) low levels of brain blood flow in aged humans and animals can lead to abnormal cell metabolism, tissue damage and memory problems independent of Abeta, 8) mild cognitive impairment can convert equally to AD or VaD, 9)and small vessel damage(including the subcellular organelles such as mitochondria) is present in the majority of AD brains after death. For this reasons, it is suggested that AD be re-classified as a oxidative stress induced vascular disorder and described as a “vasocognopathy” with the mitochondrial failure. The term aptly describes the origin of the disease(vaso:vessel blood flow), its primary effect on a system(-cogno:relating to mental ability) and its clinical course(-pathy:disorder). Re-classification of AD from a neurodegenerative to a vascular and/or mitochondrial disorder would speed the development of truly beneficial treatments or a cure, improve patient management, provide earlier diagnosis, and reduce the number of AD cases in the future by aggressively treating the risk factor that can turn on this dementia. In conclusion, a bare-bones examination of the literature reveals no compelling evidence that ABeta deposition causes AD or that it results in significant damage to brain cells. By contrast, the findings that support AD as a primary oxidative stress induced mitochondrial failure(e.g., which induces the cellular and subcellular hypoperfusion) and vascular disorder appear substantially more convincing.
Finally aging-related changes involving mitochondrial dysfunction are critical to our understanding of Alzheimer pathobiology and energy homeostasis. With age and diabetes, antioxidant enzymes are dimished and this sets the stage for future disease processes. Understanding the attenuation or loss of these critical defense mechanisms will help in our fight against this devastating disease.
We are ushering in a new approach to AD prevention and treatment through increasing these defenses with nutritional and antioxidant approaches as part of an early intervention and prevention strategy.

L6. The cerebellar cortex in Alzheimer's disease

S. J. Baloyannis.
Department of Neurology, Laboratory of Neuropatholgy, Aristotelian University
Thessaloniki, Greece.

We attempted to describe the morphological alterations of the cerebellar cortex in Alzheimer's disease, emphasizing the synaptic alterations between the climbing fibers and the Purkinje cell dendrites using silver impregnation techniques and electron microscopy, in ten cases who fulfilled all the clinical, neuropsychological and neuroimaging criteria of Alzheimer's disease. Climbing fibers are an axonic system, which projects impulses from the neuronal networks of the olivary nuclei to Purkinje cell dendrites in the molecular layer of the cerebellar cortex. No interneuron intervenes between the climbing fibers and Purkinje cells, thus the circuit may be realized as a model of very closed and direct system of neuronal interaction. A marked decrease of the number of climbing fibers was noticed in the cerebella of the patients who suffered from Alzheimer's disease in comparison with normal controls. The poverty of the climbed fibers was particularly obvious in the vermis and the floccules of the cerebellum. The majority of the synapses between the axonic terminals of the climbing fibers and the dendritic spines of the Purkinje cells demonstrated dilatation of the presynaptic component, marked poverty in synaptic vesicles and mitochondrial alterations. The postsynaptic component demonstrated mitochondrial alterations, dilatation of the cisternae of the smooth endoplasmic reticulum as well as alterations of the spinal apparatus. We may conclude that the morphological and morphometric alterations of the climbing fibers plead in favor of the involvement of the short and oligosynaptic neuronal circuits in the pathological processes of Alzheimer's disease.

L7. Screening the metabolic causes of dementia: beside Alzheimer's disease

A. Federico
Dept. Neurological, Neurosurgical and Behavioural Sciences, Medical School, University of Siena, Siena, Italy

The development of the new biochemical and molecular genetic techniques gave a great development to the understanding many causes of dementia, with the recognition of the biochemical and molecular pathogenesis of many conditions.
We will review the metabolic causes of dementia, different from Alzheimer 's disease and we will distinguish forms in which the brain dysfunction may be related to

a) changes in the internal compartment of the cells, with storage of toxic material (lysosomal diseases, ceroid lipofuscinoses);
b) changes in the energy metabolism (mitochondrial disorders)
c) changes in the metals transport (Wilson's disease)
d) changes in the physic-chemical structure of the neuronal and glial plasma membranes and canges in transport and metabolism of lipid (adrenoleucodystrophy, cerebrotendinous xanthomatosis, Nieman Pick type C)
e) changes in DNA repair (Xeroderma pigmentosum and Werner syndrome)
f) changes to vitamin adsorption and transport
g) others.

We will describe the main phenotype and the clinic-biochemical flow chart for diagnosis. We will also discuss some possibility of treatments, that will directly improve the biochemical and clinical functions.

L8. Endogenous neuroprotection and neurodegenerative disorders

László Vécsei
Department of Neurology, Faculty of Medicine, Albert Szent-Györgyi Clinical Center, University of Szeged, Szeged, Hungary

There is appreciable evidence that the neurodegeneration in Alzheimer's disease (AD) is mediated, at least partly, by neurotoxic products of the kynurenine (KYN) pathway. Possible therapeutic approaches could be to reduce the expression of these neurotoxic agents or to increase the production of putative neuroprotectant kynurenic acid (KYNAC) or make use its analogues. However, the specific involvement of the kynurenine pathway in AD, it also to be emphasized that neurodegenerative diseases share several common features. Among other common mechanisms the shift in the KYN pathway seems to be general over different neurodegenerative diseases and such, neuroprotective therapies influencing KYN pathway may be beneficial in several neurological pathologies. 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.

References:

Kincses, Zs.T., Toldi, J., Vécsei, L.: Kynurenines, neurodegeneration and Alzheimer's disease. J. Cell. Mol. Med. 14:2045-2054, 2010.

Sas, K., Robotka, H., Toldi, J., Vécsei, L.: Mitochondia, metabolic disturbances, oxidative stress and the kynurenine system, with focus on neurodegenerative disorders. J. Neurol. Sci. 257:221-239, 2007.

Sas, K., Párdutz, Á., Toldi, J., Vécsei, L.: Dementia, stroke and migraine. Some common pathological mechansims. J. Neurol. Sci. 299:55-65, 2010.

L9. Cognitive Dysfunction in Multiple Sclerosis

Panagiotis Papathanasopoulos
Professor of Neurology, Director of the Department of Neurology, University of Patras Greece

Cognitive impairment is common in multiple sclerosis (MS), occurring at all stages of the disease, even at the earliest, and can be a major source of disability, social impairment, and impoverished quality of life. Cognitive dysfunction is mainly focused on working memory, verbal fluency, speed of information processing, attention and executive function. Measures of information-processing speed appear to be the most robust and sensitive markers of cognitive impairment in MS patients. Cognitive testing in MS patients is complex and cognitive screening tests are time- and cost-saving test instruments. A comprehensive and sensitive cognitive test procedure should be administered to detect cognitive dysfunction, and recent studies demonstrate that single, predominantly speed-related cognitive tests may be superior to extensive and time-consuming test batteries in screening cognitive decline. Additional clinical factors, including disease course, fatigue, and affective disturbance, can impact the degree of MS-related cognitive impairment.

Despite weak correlation with disease duration and physical disability status, the degree of cognitive impairment in MS has been related to the extent of topographically specific neuronal tissue damage and loss. The burden of MRI-visible lesions does not fully account for the degree of MS-related cognitive impairment. These findings highlight the need to screen for cognitive deficits in MS patients to conduct potential cognitive rehabilitation intervention.

In this presentation we review studies on cognitive deficits and cognitive effects of pharmacological treatments in MS. There is evidence for a possible beneficial effect of immunomodulatory treatments, particularly of interferons, and also of acetylcholinesterase inhibitors on cognition in MS, which, however, requires evaluation in larger, multi-centre, longitudinal studies.

L10. Is there a need to redefine Parkinson's disease?

Amos D. Korczyn
The Sieratzki Chair of Neurology, Sackler School of Medicine Tel-Aviv University, Ramat-Aviv, Israel

Parkinson's disease (PD) has initially been described as a clinical syndrome, although the exact definition has changed over the past centuries. The inclusion of the pathological changes added another level of complexity, with Lewy bodies, synuclein deposits and neuronal loss in the substantia nigra being used alternatively. A third level of complexity was added with the recognition of genetic mutations resulting in parkinsonism, sometimes with and sometimes without Lewy body deposition, and the identification of frequent additional important pre-motor manifestations.

These different points of view on the definition of PD have important implications on the study of the etiology and even the therapy of PD.

L11. The limits of evidence based medicine in neurorehabilitation

Dafin F. Muresanu
Department of Neurology, University CFR Hospital, Faculty of Medicine, University of Medicine and Pharmacy “Iuliu Hatieganu ” Cluj -Napoca, Romania

The last decade has been very fertile in the development of both neurosciences and evidence based medicine (EBM), even if sometimes they did not go hand in hand.

Evidence based medicine is a concept that tries to build clinical decisions based on empirical knowledge collected from randomized control trials (RCTs). RCTs were designed in order to avoid systematic sampling errors.

Randomised controlled trials (RCTs) and systematic reviews are the most reliable methods of determining the effects of treatment.

Clinicians have to make decisions about individuals, and how best to use results of RCTs and systematic reviews. This has generated considerable debate.

They key issue remains to what extent the overall results of trials can properly inform decisions at the bedside or in the clinic.

RCTs must be internally valid (i.e., design and conduct must keep to a minimum the possibility of bias), but to be clinically useful the result must also be relevant to a definable group of patients in a particular clinical setting; this is generally termed external validity, applicability, or generalisability.

As we can see from the genomic, transcriptomic and proteomic studies of post lesional regulations, the biological reality of the nervous system is extremely complex and rather individualistic (neurotrophicity, neuroplasticity and neurogenesis responses).

Therefore, due to patients' heterogeneous responsivity in clinical practice, the approach of neurorehabilitation should be more individualistic, with better chances to manage complex situations.

This presentation will analyze the concepts of internal and external validity of RCTs and why RCTs model is difficult to be applied to neurorehabilitation clinical trials.

L12. War is Hell!

James F. Toole, MD, LLB, FRCP

*I*t is a tragedy that so many wars are caused by conflicting social religious beliefs of unwise leaders.

Because of the worldwide distribution of weapons of mass destruction, society is at a cross-over point where policies motivated by religion and/or greed lead to mass destruction.

I will consider aspects of this topic.

L13. From Diagnostics to Therapy

Konrad Maurer

Electroencephalographic and neuroimaging procedures have been used for a long time for diagnostic reasons. In the case of Motoric Evoked Potentials (MEP), where magnetic stimulation is used to investigate the motoric pathway, at the same time repetitive magnetic stimulation can be used for therapeutic reasons.
The repetitive transcranial magnetic stimulation (rTMS) can be applied in a lot of psychiatric conditions, often as "add-on"-Therapy, in otherwise difficult to treat disease entities.
Methodological procedures will be explained including details about the Navigated-rTMS, which enables to focus the magnetic beam
.
The therapeutic application and value will be shown in psychiatric conditions such as:
Auditory Hallucinations - Tinnitus - Craving - Dementia - Posttraumatic Stress Disorder - Pathological Jealousy - Chronic Pain Disorders.The therpeutical outcome will be documented by fMRI-measurements before and after treatment
.
Additionally to magnetic stimulation a new psychotherapeutical procedure will be presented, the so called "Imaginative Resonance Training -IRT", a form of psychotherapy, working with imagination. In cases with amputees`phantom pains the akefull conditions could be eliminated by IRT. The IRT was also helpful and efficacious in persons suffering from Body Identity Integrity Disorder (BIID).

L14. Social participation of the mentally and physically severe disabled - a contradictoriness of terms?

Klaus von Wild, MD. PhD, Prof. of Neurosurgery, Medical Faculty Münster, Germany

Objective: In following the Helsinki Declaration of the World Medical Association, published in 1964 and its numerous amendments, doctors are obliged to use all available means to help their patient and to leave nothing untried. There is main demand which is based on a broad consensus among politicians, doctors, representatives of religious communities ,social health care personal , rehabilitation professionals, and social workers that medical treatment interventions should be followed long term neurorehabilitation measures as to guarantee social re-integration if any possible. However, despite all efforts and new modern technologies and treatment modalities, improved rescue and care facilities some few diseases will end with a severe physical and or mental cognitive disability of the dependent human being. Living in the wealthy part of our economically orientated European world an appropriate caring at least and continuous nursing , including physical and mental stimulation modalities, has to provide for those suffering including support the relatives.
Methods: *The Standard Rules on the Equalization of Opportunities for Persons with Disabilities* were adopted by the United Nations General Assembly, forty-eighth session, resolution 48/96, annex, of 20 December 1993. Although not a legally binding instrument, the Standard Rules represent a strong moral and political commitment of Governments to take action to attain equalization of opportunities for persons with disabilities. The rules serve as an instrument for policy-making and as a basis for technical and economic cooperation. Only recently the ***UN Convention on the Rights of Persons with Disabilities*** was accepted and signed by a number of European parliaments like Germany. Above all is the ***Article 10 - Right to life.*** States Parties reaffirm that every human being has the inherent right to life and shall take all necessary measures to ensure its effective enjoyment by persons with disabilities on an equal basis with others. ***Article 17*** - Protecting the integrity of the person.Every person with disabilities has a right to respect for his or her physical and mental integrity on an equal basis with others. Article 26- Habilitation and rehabilitation.

1: States Parties shall take effective and appropriate measures, including through peer support, to enable persons with disabilities to attain and maintain maximum independence, full physical, mental, social and vocational ability, and full inclusion and participation in all aspects of life 2. States Parties shall promote the development of initial and continuing training for professionals and staff working in habilitation and rehabilitation services.3. States Parties shall promote the availability, knowledge and use of assistive devices and technologies, designed for persons with disabilities, as they relate to habilitation and rehabilitation. Article 28 focuses on the adequate standard of living and social protection .a.To ensure equal access by persons with disabilities to clean water services, and to ensure access to appropriate and affordable services, devices and other assistance for disability-related needs; b.To ensure access by persons with disabilities, in particular women and girls with disabilities and older persons with disabilities, to social protection programmes and poverty reduction programmes; c.To ensure access by persons with disabilities and their families living in situations of poverty to assistance from the State with disability-related expenses, including adequate training, counseling, financial assistance and respite care; d.To ensure access by persons with disabilities to public housing programmes; e.To ensure equal access by persons with disabilities to retirement benefits and programmes.
Results: It will be a long way to go, before all parties that become involved in caring of severely disabled human beings will accept the UN convention as to feel personally responsible, independent from his or her profession. In this respect it seems to me most important to raise awareness of the basic needs and possibilities (Article 8) by States Parties to undertake to adopt immediate, effective and appropriate measures as to raise awareness throughout society, including at the family level, regarding persons with disabilities, and to foster respect for the rights and dignity of persons with disabilities.
Conclusion; Amelioration of human beings suffering from non-treatable neurological and mental-cognitive and behavioral disabilities is now regulated. However, this needs to raise awareness of the basic needs and possibilities as a modern challenge for the social community of our times. This can be achieved when we learn to listen to the disabled persons wishes and needs and will respect his or her feelings, emotions, social- cultural and religious tradition and the given economic background.

L15. Retinal Vasoreactivity as a Marker for Cerebral Vessel Disease in Type II Diabetes?

Kerstin Bettermann, MD, PhD, Department of Neurology, Penn State College of Medicine, Hershey, PA, USA

The cerebral vasculature cannot be easily assessed in a non-invasive manner. As the eye and the brain share embryological, anatomic and physiological similarities, studies of retinal blood vessels may prove useful as a surrogate marker for cerebrovascular disease. In epidemiological studies abnormal retinal Arteriovenous Ratios (AVR) have been shown to indicate risk of stroke and cerebrovascular disease, but the association between retinal vasoreactivity measurements and cerebral blood vessel function remains unknown. An attenuated retinal vasoreactivity may indicate endothelial dysfunction in the eye and brain and may prove to be useful as a marker of cerebrovascular disease in high risk populations such as in diabetics.
STUDY GOALS: To examine **1)** the impact of diabetes at different disease stages on measures of cerebral microvascular disease and **2)** the relationship between retinal blood vessel reactivity, retinal AVRs and measures of cerebrovascular function.
METHODS: Cohort study of 19 type 2 diabetics, 3 pre-diabetics, and 8 healthy controls (ages: 37 to 74 years). Retinal vasoreactivity was measured with the Dynamic Vessel Analyzer (Imedos, Jena, Germany) following high frequency flicker light stimulation. Middle cerebral artery (MCA) flow was measured using Transcranial Doppler Ultrasound (Siemens, USA).
RESULTS: Across all groups an attenuated retinal arterial and venous light flicker response is associated with an increase in MCA RI (r=-0.41,p=0.019 artery, r=-0.39,p=0.02 vein, CI 95%) and PI (r=-0.45, p=0.01 artery, r=-0.45,p=0.009 vein, CI 95%) and a decrease in AVRs (r= 0.38, p=0.02 artery, r= 0.34, p=0.045 vein, CI 95%).
CONCLUSION: Although the study population is relatively small, there seems to be a moderate correlation between retinal vasoreactivity and measures of cerebral microvascular disease, possibly indicating that the eye reflects changes in cerebral blood vessels and stroke risk.

L.16 Leukoaraiosis and its haemodynamic causes

Kalvach P, Peisker T, Keller J, Bartoš A.
Department of Neurology, Charles University, 3rd Medical Faculty, Prague
Department of Radiology, Hospital Na Homolce, Prague

Progressive rarefaction of cerebral white matter, usually associated with atrophy, lacunes, dementia and silent brain infarctions is a common fate of aging brains. Among the processes, responsible for leukoaraiosis vascular deposits of fibrohyaline, thickening of the microvascular wall, its endothelial damage, amyloid angiopathy and blood-brain-barrier permeability are intensively studied. The dominant role is however ascribed to the haemodynamic disturbances, mostly arterial, but eventually also of venular character. Intermittent failures of cerebral blood flow are considered to cause neuronal and astrocytic swelling, oedema of both intra- and extracellular type and separations of myelin from axolemma.

Our presentation will recapitulate disturbances of cerebrovascular reactivity, due to the rigidity of the middle calibre arteries, due to improper sympathetic and parasympathetic innervation and othostatic hypotension. Results of studies revealing diminushion of the cerebral reserve for vasodilatation and constriction will be demonstrated. What our own studies have shown will be completed by examples of more sophisticated measurements, using diffusion weighting, blood oxygenation, anisotropy and susceptibility magnetic resonance investigations.

L17. Depression in Parkinson's disease: evaluation, clinical correlates and treatment

Sevasti Bostantjopoulou, M.D., Ph.D
3rd University Dept. of Neurology, Aristotle University of Thessaloniki

Depression is the most common neuropsychiatric disorder in Parkinson's disease (PD) but it is often underdiagnosed because the symptoms of PD overlap with the symptoms of depression. Prevalence rates of depressive disorders in PD range from 2.7% to 90%. Depression may occur at any time during the course of the disease, however depressive symptoms may precede the development of motor symptomato- logy, representing the first manifestation of PD in some patients.The severity of depression ranges from dysthymia to a major affective disorder. The DSM-IV criteria for depressive disorder is the current gold standard for establishing the diagnosis of depression in PD. The Beck Depression Inventory and the Montgomery-Asberg Depression Rating Scale are appropriate for screening for depression and for asses-sing the severity of depression. Attempts have been made to correlate depression with clinical parameters of the disease, such as gender, age ,age at disease onset, disease duration, motor features, stage of the disease and motor disability. Depression in PD contributes to significantly worse quality of life, increased motor-related disability, impaired cognitive function and caregiver burden. There are few controlled randomi- zed studies evaluating the efficacy of antidepressants in PD. Selective serotonin reuptake inhibitors and tricyclic antidepressants are the two major categories of antidepressants used for treating depression in Parkinson's disease.

L18. Nanostructured Li-batteries in Deep Brain Stimulation

Katerina E. Aifantis
Lab of Mechanics and Materials and 1st Neurology Clinic, Aristotle University, Thessaloniki , Greece
Physics, Michigan Technological University, Houghton, USA

The emerging field of nanotechnology has many future promising applications in the field of medicine, from cancer detection with quantum dots and cancer treatment with nanoparticles to deep brain stimulation with nanostructured Li-ion batteries. Pacemakers that are used in deep brain stimulation use a non-rechargeable Li battery that needs to be replaced quite frequently (about twice a year), since current rechargeable Li batteries cannot be used in the human body due to safety issues. The present talk after giving an overview of deep brain stimulation, and the various diseases it can treat, such as depression, tremor, chronic pain will describe next generation rechargeable Li-ion batteries that employ nanostructured materials, which allow them not only to be safe for medical applications, but also significantly smaller in size.

L19. Can Magnetic Stimulation Can Modulate Seizures in Epileptic Patients?

Photios Anninos
Department of Medicine, Democritus University of Thrace
Alexandroupolis, Greece

Abstract: The aim of this study is to investigate the influence of external magnetic stimulation (EMS) in epileptic patients using magnetoencephalographic (MEG) measurements and non-linear analytic techniques. The examined group consisted of 15 men aged 19-56 years (mean: 39.5±11.3) and 15 women aged 15-53 years (mean: 36.7±11.4). For each patient the magnetic activity was recorded from 32 points for each temporal lobe. External magnetic stimulation (EMS) with proper field characteristics (intensity: 1-7.5 pT, frequency: the **α**-rhythm of the patient (8-13Hz)) was applied in the frontal, occipital, temporal lobes and vertex for 2 minutes and the emitted brain magnetic activity was recorded again. In order to investigate if there is any alteration in the MEG complexity underlying the neural dynamics characterizing the brain before and after the EMS, chaotic analysis approach was applied for the estimation of the dimensional analysis of the existing strange attractors. The application of EMS resulted in rapid attenuation of the MEG activity of epileptic patients. The obtained results of the dimensionality calculation provide a shift from lower to higher dimensional values. Such a shift is an indication that we are dealing with a chaotic system similar with the one characterizing normal subjects. The increased values of the dimensional complexity and the lower activity of the MEG after the application of EMS strongly supports the beneficial effects of EMS in epileptic patients.

L20. The influence of internal secretion in Multiple Sclerosis

Euphrosyni S. Koutsouraki
Assistant Professor of Neurology
1st Department of Neurology, Aristotelian University
Thessaloniki, Greece

The classic role of internal secretion (hormones) is to promote normal growth, metabolism and reproduction. Hormones are able to initiate a cascade of responses in their target tissues that is often followed by a feedback loop to bring physiological systems back to equilibrium. They come in five major classes: steroids, amino acid derivatives, small neuropeptides, large proteins and vitamin derivatives. Two major observations led to the hypothesis that hormones influence autoimmune diseases. First, corticosteroids influence inflammation, and many autoimmune disorders respond to corticosteroids. Second, most autoimmune diseases have a gender bias and are more common in women. In addition some autoimmune disorders improve, while others worsen, during periods of significant hormonal change such as pregnancy.

While multiple sclerosis (MS) may not be an autoimmune disease, it has many features in common with autoimmune diseases including the gender bias and tendency to relapse and remit, and immunomodulation appears to be at least somewhat effective in containing the inflammatory component of the disease process.

The function of hormones has expanded to include immunomodulation and neuroprotection in addition to their classic roles. The story of how hormones influence inflammation and neuron and glial function is being slowly unraveled. There is increasing evidence that estrogen, progesterone and testosterone contain immune responses and influence damage repair in the nervous system. Hormones such as prolactin and vitamin D are being explored as immunomodulators and may influence diseases such as MS or maybe used therapeutically to modulate the immune response. More recently identified hormones, such as leptin and gherlin, may also influence the course of disease.

Research exploring how internal secretion influences the immune system began in the 1950s and 1960s. These studies have unquestionably changed our thinking about hormone action. In the last decade, there has been a tremendous growth in our understanding of the non classical immunomodulating role of hormones. Their cytokine-like effects on the cells of the immune system and their cytoprotecive or cytotoxic effects on cells of the nervous system are particularly relevant to MS. Recent studies have revealed that some hormones can be produced in immune cells themselves. Hormone receptors exist on or in immune cells as well as in cells of the CNS. Internal secretion has an expansive effect on a wide variety of cells.

L21. The role of cerebellum in the pathophysiology of dystonia

Marianthi Arnaoutoglou, MD, PhD
Assis. Professor of Neurology
1st Department of Neurology
A.H.E.P.A Hospital, AUTH

Dystonia is a syndrome of sustained and simultaneous muscular contraction of agonists and antagonists leading to twisting, abnormal posture and repetitive movements. During voluntary movement we may observe contraction of adjoining and distal muscles (overflow) or rhythmic muscular contractions leading to tremor

The pathophysiological mechanisms are quite obscure. In the last years, three hypothesis have been implemented: a) Hyperexcitability Loss of inhibition, leading to abnormal excessive output

b) Abnormal neuronal plasticity and c) somatosensory alterations of neuronal circuitry. In addition, the anatomical localization of cerebral dysfunction in dystonia is still a riddle. Several anatomical sites have been proposed: the supplementary motor cortex , the somatosensory cortex, proprioceprive receptors, the basal Ganglia the brainstem e.t.c. Recently, there is new evidence (clinical and experimental) supporting the role of cerebellum in the pathophysiology of dystonia.

Several **structural imaging studies** link defects of the cerebellum or its connections with dystonia. Additionally, surgical studies have shown some patients with dystonia respond to **DBS of the ventral posterior thalamus** or targeted region of the area, which is the **major recipient of cerebellar afferents** and it is not an integral part of the basal ganglia.

Also, well documented **animal studies** show that ablation of the cerebellum lead to ataxia while pharmacological excitation causes dystonia.

L22. Indices of carotid plaque instability in asymptomatic individuals based on transcranial doppler

T. J. Tegos, Assistant Professor of Neurology,
A Department of Neurology, AHEPA Hospital, Aristotelian University of Thessaloniki, Greece

The characteristics of the unstable carotid plaque on cervical ultrasound have long been recognized. Recent research interest has focused on the characteristics of intracranial circulation in the presence of an unstable carotid plaque and more specifically on cerebral collateral circulation and presence of intracranial emboli, all evaluated by means of transcranial Doppler.

Cerebral collateral circulation can be evaluated by means of invasive methods (1) or non-invasively by means of either the administration of CO_2 (by mask), or intravenous administration of acetazolamide or Breath Holding Test. The common denominator of the latter is the vasodilation of peripheral cerebral circulation. Based on the above mentioned non-invasive methods, reduced cerebrovascular reactivity has been shown in cerebrovascular accidents and inadequate cerebral collateral circulation but also in critical carotid stenosis, cerebral small vessel disease, increased blood viscosity and increased intracranial pressure.

Previous research has demonstrated that reduced cerebrovascular reactivity at baseline (based on the aforementioned noninvasive methods) could predict the development of a neurovascular event in asymptomatic individuals with carotid disease (2, 3, 4).

Emboli detected on transcranial Doppler in the ipsilateral to an asymptomatic carotid plaque middle cerebral artery have a predictive value for the development of a neurovascular symptom. This position has reached statistical significance in some studies (5, 6, 7, 8) and just a statistical trend in one (9). The final outcome of the Asymptomatic Carotid Emboli Study is awaited (10).

Based on the above mentioned studies it might be suggested that there is not an unstable carotid plaque as such, but an unstable system consisting of a carotid plaque and the cerebral vasculature. It is hoped that by pursuing this avenue the problem of the asymptomatic carotid plaque can be solved with a decent degree of certainty.

REFERENCES

- Tegos TJ, Sabetai MM, Robless P, Kalodiki E, Bassett P, Stansby G, Nicolaides AN. The significance of the cerebral collateral capacity in patients with carotid atheroma. Eur J Vasc Endovasc Surg 2000 Nov;20(5):434-40
- Gur AY, Bova I, Bornstein NM. Is impaired cerebral vasomotor reactivity a predictive factor of stroke in asymptomatic patients? Stroke 1996; 27: 2188-2190
- Silvestrini M, Vernieri F, Pasqualeti P, Matteis M, Passarelli F, Troisi E, Caltagirone C. Impaired cerebral vasoreactivity and risk of stroke in patients with asymptomatic carotid artery stenosis. JAMA 2000: 283: 2122-2127
- Markus H, Gullinane M. Severely impaired cerebrovascular reactivity predicts stroke and TIA risk in patients with carotid artery stenosis and occlusion. Brain 2001: 124: 457-467
- Siebler M, Nachtmann A, Sitzer M, Rose G, Kleinschmidt A, Rademacher J, Steinmetz H. Cerebral micoembolism and the risk of ischemia in asymptomatic high grade internal carotid artery stenosis. Stroke 1995: 26: 2184-2186
- Molloy J, Markus HS. Asymptomatic embolization predicts stroke and TIA risk in patients with carotid artery stenosis. Stroke 1999; 30: 1440-1443
- Orlandi G, Fanucchi S, Sartucci F, Muri L, Liapis CH, Kakisis JD, Kostakis AG. Can microembolic signals identify unstable plaques affecting symptomatology in carotid stenosis? Response. Stroke 2002; 33: 1744-1746
- Spence DJ, Tamayo A, Lownie SP, Ng WP, Ferguson GG. Absence of microemboli on transcranial Doppler identifies low risk patients with asymptomatic carotid stenosis. Stroke 2005; 36: 2373-2378
- Abbott AN, Chambers BR, Stork JL, Levi CR, Bladin CF, Donnan GA. Embolic signals and prediction of ipsilateral stroke or transient ischemic attacks in asymptomatic carotid stenosis. Stroke 2005; 36: 1128-1133
- The Asymptomatic Carotid Emboli Study: study design and baseline results. International Journal of Stroke 2009; 4: 389-405

L23. Carotid plaque echostructure: does it play a role in clinical decision making?

Athanasios D. Giannoukas, MSc, MD, PhD, FEBVS, Professor of Vascular Surgery
Faculty of Medicine, University of Thessalia, Larissa, Greece

All the existing so far randomised trials estimating the risk of stroke have been solely based on the degree of luminal stenosis. This remains the main risk so far factor but research in the field has implicated that there might be other factors that could play an additive role in the risk of stroke. One of these is the morphology of the carotid plaque that has attracted special interest. Ultrasonographic and recently MRI and CT analysis of the plaque structure have triggered the research. The fact that high resolution ultrasonography, an inexpensive, easily repeatable and free of side effects method, has contributed seriously in the assessment of the carotid plaque. Several studies have shown that totally or predominantly echolucent plaques and plaques with high heterogeneity in combination with high grade stenosis (> 60-70%) are those related more often with neurologic events. Also, plaques in which their echolucent areas are close to the luminal surface of the plaque are potentially more dangerous. All these findings have enabled the researchers to produce models and cut-off points that may be applicable in clinical practice. One of these is the grey-scale median (GSM) that is produced by special computer-based software analysing the pixels of the ultrasonic image of the carotid plaque. A cut-off point of GSM ≤ 25 has been used in order to identify those plaques that be associated with more neurologic events. This cut-off point has been proposed, based on the results of the ICAROS study that it should be taken into account in the decision making regarding the treatment. In plaques with GSM ≤ 25 stenting is not advisable even if a brain protection is used.
The plaque echomorphology has been also used in the estimation of risk of stroke in asymptomatic high degree carotid stenosis in an attempt to identify a subgroup of asymptomatic patients with an annual risk of stroke ≥ 4% in whom intervention would offer a clear benefit if used in addition to best medical management. The recent report of the ACSRS study has shown that plaque echomorphology is one among other factors that help in the identification of a high risk group of asymptomatic patients. Nevertheless, further natural history studies are needed for the prospective validation of these results.

L24. Myasthenia gravis: recent developments on its laboratory diagnosis and antigen-specific therapy

S. Tzartos1,2, M. Belimezi, A. Kokla, G.Kordas2, G. Lagoumintzis2, K. Lazaridis1, K. Poulas2, A. Sotiriadis1, N. Trakas1 and P.i Zisimopoulou1
1Department of Biochemistry, Hellenic Pasteur Institute, Athens, Greece 2Department of Pharmacy, University of Patras, Patras, Greece

Myasthenia gravis (MG) is a model autoimmune disease due to the very well characterized auto-antigen(s) and the relatively simple pathogenic mechanisms. It may be generalized or, less frequently, purely ocular. In about 85-90% of the patients with generalized MG and 50% of those with ocular MG it is due to autoantibodies against the skeletal muscle acetylcholine receptor (AChR). These autoantibodies cause destruction of the AChRs, thus inhibiting neuromuscular transmission, but the inducing mechanism of these autoantibodies is still unknown. In about 6% of the generalized MG patients, the disease may be due to autoantibodies to the protein kinase MuSK, which contributes to AChR clustering in the neuromuscular junction. The target of the autoantibodies of the remaining MG patients is still unknown. Many MG patients have antibodies against titin and ryanodine receptor, with prognostic value for the presence of thymoma.
Anti-AChR and anti-MuSK antibodies are usually detected by radioimmunoassays. Although these approaches are quite sensitive, they may do not detect such antibodies in the sera of patients whose most produced antibodies are immobilized on the muscle embedded antigens. We are developing highly sensitive radioimmunoassays to overcome this problem.

Current treatments for MG are non-specific, causing several side effects, and are of moderate effectiveness. Several international efforts are currently been made to identify the triggering MG mechanisms and to selectively interfere with the autoantibody production in MG, ideally permanently. We aim at a less ambitious and temporary, yet antigen-specific and probably easier to establish therapeutic approach which aims at the selective removal of the already formed pathogenic autoantibodies from the patient. Specifically, we are developing an antigen-specific alternative to plasmapheresis since the latter removes indispensable plasma components, in addition to the pathogenic autoantibodies. To this aim we are expressing the extracellular domains (ECDs) of all five human muscle AChR subunits; we then immobilize the ECDs on columns and use these columns to immunoadsorb the patients' autoantibodies from their sera. These columns were capable of specifically eliminating the majority of the anti-AChR antibodies from ~30% of MG sera. Interestingly, the autoantibody-depleted sera lost their pathogenic activity (when injected into experimental rats); this proves that the anti-AChR antibodies are indeed the main pathogenic factor in anti-AChR MG and suggests that our approach is likely to be an efficient antigen-specific therapy for MG. Its further application to the therapeutic removal of anti-MuSK autoantibodies is also in progress, whereas its application to other autoantigens (like aquaporin-4) is also under consideration.

L25. Η Εταιρεία Νόσου Alzheimer και Συναφών Διαταραχών Αθηνών

Δρ. Παρασκευή Σακκά,Νευρολόγος Ψυχίατρος
Πρόεδρος Εταιρείας Νόσου Alzheimer & Συναφών Διαταραχών

Η **Εταιρεία Νόσου Alzheimer και Συναφών Διαταραχών Αθηνών** είναι μη κερδοσκοπικός οργανισμός, που ιδρύθηκε το 2002 από συγγενείς ανοϊκών ασθενών και επαγγελματίες υγείας που ασχολούνται με τη νόσο Alzheimer. Έχει σκοπό να προωθήσει την ενημέρωση, την κατανόηση και την υποστήριξη όλων όσων έχουν οποιαδήποτε σχέση με τη νόσο αυτή. Πραγματοποιεί μεγάλο αριθμό ενημερωτικών, εκπαιδευτικών και άλλων εκδηλώσεων για τους φροντιστές, τους ασθενείς, αλλά και το ευρύτερο κοινό. Σήμερα αριθμεί 1450 μέλη.

Με την υποστήριξη του Υπουργείου Υγείας και Κοινωνικής Αλληλεγγύης, η Εταιρεία Νόσου Alzheimer και Συναφών Διαταραχών Αθηνών λειτουργεί 2 Κέντρα Ημέρας για ανοϊκούς ασθενείς, στην οδό Μάρκου Μουσούρου 89 και Στίλπωνος 33, στο Παγκράτι και στην οδό Βαθέως 25 και Πανόρμου σε συνεργασία με το Δήμο Αθηναίων και τη Λέσχη Φιλίας Αμπελοκήπων).

Σε συνεργασία με το Δήμο Αμαρουσίου λειτουργεί Κέντρο Ημέρας στην οδό Ζήνωνος Ελεάτου 8 στο Μαρούσι (τηλ. 210 6180073).

Τα Κέντρα Ημέρας είναι μονάδες ημερήσιας θεραπευτικής φροντίδας ασθενών με νόσο Alzheimer και οι δραστηριότητές τους περιλαμβάνουν Ιατρείο Μνήμης, ομάδες νοητικής ενδυνάμωσης, συνεδρίες τροποποίησης προβληματικών συμπεριφορών, ατομική και ομαδική δημιουργική απασχόληση, λογοθεραπεία, εργοθεραπεία και γυμναστική.

Άλλη δραστηριότητα της Εταιρείας Νόσου Alzheimer και Συναφών Διαταραχών Αθηνών είναι το πρόγραμμα "**Φροντίδα στο Σπίτι για ανοϊκούς ασθενείς**" που απευθύνεται σε οικογένειες ανοϊκών ασθενών που δεν μπορούν να μετακινηθούν, λόγω της βαρύτητας της νόσου ή άλλων προβλημάτων. Στόχος του προγράμματος είναι η παραμονή των ανοϊκών ασθενών στο οικογενειακό περιβάλλον και η αποφυγή της ιδρυματικής φροντίδας με αποτέλεσμα την ανακούφιση του φορτίου των φροντιστών κτλ.

Η **Εταιρεία Νόσου Αλτσχάιμερ και Συναφών Διαταραχών Αθηνών** εκδίδει και διανέμει δωρεάν σε κάθε ενδιαφερόμενο πλούσιο ενημερωτικό υλικό καθώς και εφημερίδα τρίμηνης έκδοσης με τον τίτλο **Ενημέρωση για τη νόσο Αλτσχάιμερ.**

Επίσης πραγματοποιεί ένα μεγάλο αριθμό επιστημονικών εκδηλώσεων για επαγγελματίες Υγείας και εκδηλώσεις ευαισθητοποίησης για το κοινό.

Ιστοσελίδα : www.alzheimerathens.gr

Υπογράψτε την Ελληνική Πρωτοβουλία Δράσης για τη νόσο Alzheimer

POSTERS

P1. An immunohistochemical study of NMDA receptors in human cerebellum and hippocampus

E. S. Koutsouraki, J. J. Anastasiades, V.i G. Costa, S. J. Baloyannis
1st Department of Neurology, Aristotelian University ,AHEPA Hospital, Thessaloniki, Greece
Immunohistochemical Laboratory

ABSTRACT

Glutamate N-methyl-D-aspartete (NMDA) receptors comprise a family of homologous subunits, which co-assemble into hetero-oligomeric protein complexes. Based on amino acid sequence identity, these subunits fall into two major types, termed NR1, comprising eight splice variants, NR1-1a, 1b through to NR1-4a, 4b and NR2ANR2D.
The aim of our investigation was to demonstrate the wide distribution of NMDA receptors, in the human adult cerebellum as well as in hippocampus.
Human hippocampus and cerebellum were obtained at autopsy from two male individuals, aged 24 and 48 years, with no evidence of neurological or psychiatric disease, dying during a car accident and without any obvious brain injury. The brains were immersion-fixed in 10% formalin for 2 days and then embedded in paraffin wax; 5μm sections were cut, deparaffinised in xylene and rehydrated, immunostained using polyclonal NR antibodies (Anti-Rabbit Poly HRP IHC (Detection kit) and counterstained with Mayer's hematoxylin. These polyclonal antibodies were subsequently used to map the cellular distribution of the NMDA receptor subunits NR2A & NR2B.
The present immunohistochemical research of human adult cerebellum and hippocampus, using human anti-rabbit NMDA R 2A & B at the light microscope, demonstrates that the majority of neurons in the dentate gyrus, the large pyramidal neurons of the hippocampus, the granular cells of the cerebellum as well as the main cerebellar neuron, namely Purkinje cell, stained deeply by the monoclonal antibody, suggesting that the majority of the neuronal network in cerebellum and hippocampus uses as neurotransmitter the excitatory aminoacids, widely on NMDA receptors, on the postsynaptic membrane on the system of NMDA R2A & B. It's well known that many other systems of neurotransmitters are used by both of those structures of the CNS such as the GABA, the acetylcholine (Ach), the monoamines and the neuropeptides. Our findings, demonstrating that the majority of cells are stained by the monoclonal antibody related to NMDA receptors, emphasize the importance of the excitatory system of the glutamate in the cerebellum and the hippocampus, underlying the important role that this system may play in memory function and cognition and at the same time the participation of the NMDA receptors in many phenomena of excitation of the hippocampus and the cerebellum which might be related to epileptic seizures, motor and behavioral disorders.

P2. Levels of Antibodies against Gangliosides GM1, GD1b and GQ1b in Demented Patients

E.Hatzifilippou[2], E. Koutsouraki[1,2], T. Banaki[1], M.Traka[1], V.G.Costa[1,2], S.J. Baloyannis[1,2]
[1] Memory clinic of the 1st Department of Neurology, Aristotelian University
[2] Neuroimmunological Laboratory of the 1st Department of Neurology, Aristotelian University, AHEPA Hospital, Thessaloniki, Greece

ABSTRACT

The aim of our study was to evaluate the levels of anti-GM1, anti-GD1b and anti-GQ1b in demented patients, correlating them with the type and the severity of dementia.
We examined the concentrations of antibodies against the gangliosides: GM1, GD1b and GQ1b in the serum of a total 103 demented patients (68 females and 35 males) with a male to female ratio of 1:1.9 at a mean age of 71.5±8.9 years for females and 71.6±9.0 for males. Most of the patients suffered from vascular and frontotemporal dementia.

56 healthy individuals at a mean age of 71.3±8.8 years, have also been examined for the presence of antibodies.

- 66% (n=68) of the patients revealed increased values of anti-GM1 (mean 38 EU/ml±9.1), whereas 87.5% (n=49) of the healthy controls demonstrated a mean concentration of anti-GM1 IgM 13.6 EU/ml±3.6.
- Increased concentrations of anti-GD1b IgM (mean 30.6 EU/ml ±3.7) were detected in 35% of the patients (n=36), whereas 82% (n=46) of the healthy individuals demonstrated lower concentrations of anti-GD1b IgM (12.4 EU/ml±3.3).
- Only 9% (n=9) of the patients revealed high concentrations of anti-GQ1b IgM (mean 28 EU/ml±5.7). All healthy controls were negative in anti-GQ1b IgM (7.5 EU/ml±4.8).
- Patients who revealed the most increased levels of anti-GM1 IgM (>30 EU/ml, with normal values <20 EU/ml), demonstrated the lowest scores in the MMSE scala: 8-18/30 and they were aged between 69-73 years.
- Patients with higher concentrations of anti-GD1b IgM (over 21 EU/ml, with normal values <20 EU/ml), also revealed a moderate to severe dementia (MMSE 8-18/30). and their age was between 74-75 years.

Our findings are indicative of a possible correlation between the levels of anti-GM1 and anti-GD1b with the grade and the type of dementia.

P3. Broca's area in Alzheimer's disease

Ioannis A. Mavroudis[1], Luc Flavien Adipepe[1], Marina Manani[1], Samouel N. Njau[2], Vassiliki Costa[1], Stavros I. Baloyannis[1]
1.Laboratory of Neuropathology and Electron Microscopy, First Department of Neurology, Aristotelian University of Thessaloniki, Greece
2.Laboratory of Forensic medicine, Aristotelian University of Thessaloniki, Greece

Alzheimer's disease constitutes the most common cause of cognitive impairment in senium and presenium. The most typical decline of cognition concerns, global memory including semantic memory, the ability to utilize cognitive support for episodic remembering, visuospatial function, attention abstractions, language, praxis and psychomotor function.
Broca's area provides the neural circuitry for word formation. This area in combination with Werincke's language comprehension center is essential for the production and understanding of human speech.
This study is based on the morphological analysis and morphometric estimation of the neurons and vasculature of pars opercularis of the inferior frontal gyrus, from 5 healthy individuals, and 7 Alzheimer's disease patients, using Golgi method, Gallyas and Bielschowsky's techniques and Holzer staining.
A substantial loss of dendrites and dendritic spines has been noticed, while alterations of Astrocytes and morphological changes of the vasculature have also been demonstrated in Alzheimer's disease patients. Gallyas and Bielschowsky's techniques reveal deposition of senile plaques and neurofibrillary tangles mainly in Alzheimer's disease brains, while in normal aging senile plaques are seen to be close to vessels.
The morphological and morphometric study pleads in favour of the crucial role of Astrocytes and vasculature alterations in the Alzheimer's disease pathogenetic mechanism. Furthermore dendritic and spinal pathology are the most important causes of the decline of the higher mental faculties such as speech comprehension and word formation impairment.

P4. Morphological and morphometric alterations of the neurons of Edinger and Westphal nucleus in Alzheimer's disease.

D. F. Fotiou[1,2], I. A. Mavroudis[1], L.F. Adipepe[1], D. Mytilinaios[1], K. Tsamis[1], V. G. Costa[1], S. J. Baloyannis [1,2]
1. Laboratory of Neuropathology and Electron Microscopy, 1st Department of Neurology, Aristotelian University of Thessaloniki, Greece
2. Laboratory of Clinical Neurophysiology, 1st Department of Neurology, Aristotelian University of Thessaloniki, Greece

It is well known that Alzheimer's disease is characterized by severe loss of cholinergic neurons of the basal nucleus of Meynert, mainly related to the cognitive impairment and loss of professional skills.
Studies in neurophysiology using pupillometry reveals significant decrease in vMax and in AccMax during pupil miosis compared to normal individuals, a fact that pleads in favour of cholinergic loss in Edinger and Westphal nucleus.
The present study is based on the morphological and morphometric examination of neurons of Edinger and Westphal nucleus in patients suffered from Alzheimer's disease in comparison to normal aged individuals, consisting a part of a wider study of the parasympathetic nuclei of the brainstem in Alzheimer's disease.

P5. Fragmentation of nucleoli and morphological changes of the nuclei are related to neuronal loss, dendritic pathology and spinal loss in Alzheimer's disease

I.A. Mavroudis[1], D.F. Fotiou[1], M. G. Manani[1], S. N. Njau[2], V.G. Costa[1], S.J. Baloyannis[1]
1. Laboratory of Neuropathology and Electron Microscopy, 1st Department of Neurology, Aristotelian University of Thessaloniki, Greece
2. Laboratory of Forensic medicine, Aristotelian University of Thessaloniki, Greece

Alzheimer's disease constitutes the most common cause of cognitive impairment in senium and presenium. The most typical decline of cognition concerns, global memory including semantic memory, the ability to utilize cognitive support for episodic remembering, visuospatial function, attention abstractions, language, praxis and psychomotor function. From the neuropathological point of view Alzheimer's disease is characterized by neuronal loss, neurofibrillary degeneration and extracellular deposits of Aβ peptide, as well as substantial loss of dendritic spines. Furthermore changes of the mitochondrial cristae, accumulation of osmiophilic material and decrease of their size, and morphological alterations of the Golgi apparatus have also been described. The nucleolus is the most obvious structure seen in the nucleus of a eukaryotic cell when viewed in the light microscope. Consequently, it was so closely scrutinized by early cytologists that an 1898 review could list some 700 references. The nucleolus is the site for the processing of rRNAs and their assembly into ribosome subunits. Unlike many of the major organelles in the cell, the nucleolus is not bound by a membrane; instead, it is a large aggregate of macromolecules, including the rRNA genes themselves, precursor rRNAs, mature rRNAs, rRNA-processing enzymes, snoRNPs, ribosomal proteins and partly assembled ribosomes.
Ribosomal RNA genes are involved in cell transcription and translation processes and can modulate gene expression. The rRNA genes have an important role in forming the nucleolus. The size of the nucleolus reflects the number of ribosomes that the cell is producing. Its size therefore varies greatly in different cells and can change in a single cell, occupying 25% of the total nuclear volume in cells that are making unusually large amounts of protein.
In addition to its important role in ribosome biogenesis, the nucleolus is also the site where other RNAs are produced and other RNAprotein complexes are assembled (snRNP subunits). Other important RNAprotein complexes, including telomerase and the signal recognition particle are also believed to be assembled at the nucleolus. Finally, the tRNAs (transfer RNAs) that carry the amino acids for protein synthesis are processed there as well; like the rRNA genes, those

encoding tRNAs are clustered in the nucleolus. Thus, the nucleolus can be thought of as a large factory at which many different noncoding RNAs are transcribed, processed, and assembled with proteins to form a large variety of ribonucleoprotein complexes.
This study was carried out in order to figure out if there is a relationship between the morphology of the nucleus and the nucleolus and the neuronal loss, the dendritic and spinal pathology as well as the neurofibrillary degeneration in Alzheimer's disease.

P6. Vascular and blood brain barrier pathology are related to dendritic alterations and spinal loss in Alzheimer's disease.

I.A. Mavroudis[1], D.F. Fotiou[1], SN Njau[2], K. Tsamis[1], V. Costa[1], S. J. Baloyannis[1]
1. Laboratory of Neuropathology and Electron Microscopy, First Department of Neurology, Aristotelian University of Thessaloniki, Greece
2. Laboratory of Forensic medicine, Aristotelian University of Thessaloniki, Greece

Abstract
Alzheimer´s disease is a devastating disorder of the central nervous system, involving progressive cognitive impairment, characterized by profound memory loss, learning inability and behavioral changes.
The neuropathological spectrum of the disease includes neurofibrillary degeneration and deposition of senile plaques, dendritic and spinal pathology, as well as alterations of the mitochondria and the Golgi apparatus.
In this study we tried to demonstrate the morphological and morphometric changes of the vasculature in cortical and subcortical areas in normal aging and Alzheimer's disease and to correlate them to the neurofibrillary degeneration and dendritic and spinal pathology.
Fifteen brains obtained at autopsy, three from patients suffered from Alzheimer`s disease, six from normal aged individuals and six from young healthy individuals have been used. Performing neuroanatomy we excised small parts from the visual cortex, the acoustic cortex, the insula of Reil, Broca's area, Wernicke's area, the supramarginal gyrus, the superior and inferior colliculi, the medial and lateral geniculate nuclei, the mammillary bodies, the amygdaloid bodies, the cerebellar cortex and the dentate nucleus of the cerebellum. The samples were processed for silver impregnation, according to rapid Golgi and modified Golgi methods, Gallyas technique, as well as Bielschowsky' s, Nissl and Holzer staining procedures.
Neurofibrillary degeneration, synaptic loss and marked abbreviation of the dendritic arborisation in correlation with vascular pathology were seen in the majority of the neurons in Alzheimer's disease patients comparatively to normal controls. These findings suggest that morphological alterations of the vasculature play a crucial role in Alzheimer's disease pathogenic mechanism.

P7. Human Amygdaloid bodies. Cytoarchitecture and efferent fiber system.

L. F. Adipepe[1], I.A. Mavroudis[1], D. F. Fotiou[1], S.N. Njau[2], V. Costa[2], S. J. Baloyannis[2]
1. Laboratory of Neuropathology and Electron Microscopy, First Department of Neurology, Aristotelian University of Thessaloniki, Greece
2. Laboratory of Forensic medicine, Aristotelian University of Thessaloniki, Greece

Abstract
Amygdaloid bodies lie at the medial aspect of the temporal lobe. They consist of a complex of subnuclei, namely the superficial cortical nucleus, the central nucleus, the basal nucleus consisting of a parvocellular medial part and a magnocellular lateral part and the lateral nucleus. The subnuclei form two groups, the corticomedial and the basolateral group.
The most important efferent fiber system of the amygdaloid bodies is the stria terminalis, which

terminates in the septal nuclei, in the preoptic area and in the nuclei of hypothalamus.
Amygdaloid bodies are involved in emotional responses, especially in fear and fear conditioning and are seem to be behavioral awareness areas.
In this study we attempt to describe the cytoarchitecture of amygdaloid bodies and the efferent fiber system to the structures of diencephalon, using Golgi staining and Nauta method.
Three main types of cells have been noticed. The first type includes spiny cells with pyramidal-like somata with three to seven dendrites emanating from the soma. One of the dendrites is usually more prominent than the others and thus has been likened to the apical dendrite of cortical neurons. Some neurons appear to have two apical dendrites and are more like the spiny stellate cells of the cortex.
The second main group of cells has slightly smaller somata and resembles nonspiny stellate cells of the cortex. These cells have two to six primary dendrites that lack spines and form a relatively spherical dendritic field.
The third type of cells has an ovoid or fusiform soma and three to five nonspiny primary dendrites from which moderately spiny, sparsely branching secondary and tertiary dendrites arise.

P8. Cytokine profile in patients with Alzheimer's disease

M. Traka, E. Hatzifilippou, T. Banaki, E. Koutsouraki, V.G. Costa, S.J. Baloyannis.
1st Department of Neurology, AHEPA Hospital, School of Medicine, Aristotelian University, Thessaloniki, Greece.

Aims of the study: The role of chronic inflammation in the pathogenesis of Alzheimer's disease (AD) has been suggested in a plethora of studies. Cytokines are a group of heterogeneous proteins of low molecular weight with a fundamental role in the regulation of inflammatory processes. The objective of the present study is to evaluate immune alterations and immunological markers in patients suffering from AD.
Patients and methods: Five cytokines IL-2, IL-6, IL-10, IL-12 and TNF-α, were measured in the blood serum of patients with probable AD according to the NINCDS/ARDRA criteria, as well as in age-matched non demented controls. The dementia stage of the patients was determined according to the CDR scale. Cytokines were measured using the enzyme-linked immunosorbent assay (ELISA).
Results: A statistically significant decrease was observed in the level of IL-10 and IL-12 in the serum of patients with AD as compared against healthy patients. No statistically significant correlation was observed between the mean interleukin levels and the age of the patients or the stage of dementia.
Conclusion: Data suggest the existence of detectable immunobiological alterations in AD and confirm, in addition to the progressive loss of neuronal synapses, neurons and neuronal functions, the participation of the immune system in clinical manifestations and probably in the pathogenesis of the disease.

P9. Cognitive assessment in alcohol-dependent patients and patients with Alzheimer's disease: The distinct neuropsychological profile

Theotoka I, Kapaki E, Ilias I, Paraskevas GP, Liappas I.
Department of Psychiatry and Neurology, Athens National University, School of Medicine, Eginition Hospital, Athens, Greece.

BACKGROUND: Heavy and chronic alcohol dependence and Alzheimer's disease may share some neuropsychological characteristics.
PATIENTS AND METHODS: The pattern of neuropsychological characteristics of 33 alcohol-dependent patients who reported memory disturbances were evaluated and compared to the

neuropsychological performance of 38 patients with mild-stage Alzheimer's disease and 73 healthy subjects, serving as controls. Alcohol-dependent patients were examined with tools concerning the pattern of alcohol abuse and problems related to alcohol consumption. All groups completed a full battery of neuropsychological tests for the assessment of cognitive functions, such as different kinds of memory, attention, executive function etc.
RESULTS: Alcohol-dependent patients fared worse compared to the control subjects in every test used. The comparison of alcohol-dependent patients versus patients with Alzheimer's disease showed that the latter are much more burdened, as far as cognition is concerned, in all aspects of memory.
CONCLUSION: Alcohol-dependent patients, even if they are not demented, have mild cognitive impairment in all domains of cognition (memory and frontal functions) in comparison with controls which performed within the norms. Verbal fluency, working memory and frontal functions were impaired at the same degree in alcohol-dependent patients and in patients with Alzheimer's disease. Memory problems were more pronounced in Alzheimer's disease patients.

P10. Functional Status of Brain Hemispheres in Amnestic Mild Cognitive Impairment (aMCI)

Fereshteh Sedaghat[1,2], Amin Rakhshani[3], Thomas Tegos[1], Stavros J Baloyannis[1]
[1]Aristotle University of Thessaloniki,[2] Nuclear Medicine Research Center- Mashhad University of Medical Sciences- Iran,[3]Depatrment of Computer Science, University of Crete, Greece

Objective: To demonstrate functional status of brain in cases with aMCI.
Patients and Methods: Twenty persons with a chief complaint of forgetfulness were tested by “Sedaghat et al. memory test” (a PC-based simply applied memory and attention test). Eleven cases showed abnormal memory score (lower than 1.5 Z score). They were followed-up for 8 months. Only 6 of them showed a decline in their memory status in this period. These 6 cases underwent brain perfusion scan using HMPAO single photon emission computed tomography (SPECT).
Results: Brain SPECT showed blood hypoperfusion in left hemisphere comparing to right. Precuneus showed hypoperfusion in all these 6 cases.
Conclusion: Left brain hemisphere and precuneus may be the first regions affected in very early stage of memory deficit.

P11. The diagnosis of cognitive impairment in parkinson disease

Dr. I. THEOTOKA, Clinical Neuropsychologist, Department of Psychiatry, Athens University Medical School, “Eginition” Hospital

In this presentation, we will discuss the neuropsychological assessment of dementia in Parkinson's disease. The aim of the neuropsychological assessment are the evaluation of cognitive deficiency, the differential diagnosis, the assessment of severity of illness, the creation of a rehabilitation plan, the evaluation of therapeutic outcome and the family education. The diagnostic and therapeutic demand must be clear. It is also important to clear the right criteria, in order to use the appropriate scales.
We will refer to valid and sensitive scales for the every day practice and the research. We will discuss also the advantages and disadvantages of these tools, in order to select the appropriate ones, considering the frame of the patient and the therapeutic team.
We will also present the validation in greek population of a new neuropsychological tool (PANDA questionnaire) for the assessment of cognitive deficiency and dementia in Parkinson's disease.

P12. Epidemiologic data of Mutliple Sclerosis in Northern Greece during the last thirty years (1979-2008)

E.S. Koutsouraki, A. Fotakidou, M. Arnaoutoglou, T. Gatsios, S. Koukoulidou, V.G. Costa, S.J. Baloyannis
1st Department of Neurology, AHEPA Hospital, School of Medicine, Aristotelian University, Thessaloniki, Greece

Abstract

We analysed retrospectively 1180 MS (multiple sclerosis) patients admitted in the 1st Department of Neurology of Aristotelian University, AHEPA Hospital, Thessaloniki, Greece, during the years 1979-2008. We selected those patients with a definite MS diagnosis according to the criteria of Poser; retrospective application of the McDonald's criteria was included.

The aim of the present study was to estimate the incidence of the disease, the female predominance, the seasonal and geographic distribution and to present the initial symptoms, the risk factors and the familial cases.

It was demonstrated an average annual rate of 39 MS patients, and a female to male ratio of 1.6:1. This ratio is similar with the ones presented in earlier studies in northern Greece. Our study indicates, similar to other studies, a gradual increase of the incidence of MS, probably due to improved diagnostic approaches or to changes in the way of living. The highest incidence during the period 1979-2008, according to our data, occurred in 1998 and the lowest in 1980.

The mean age during the exacerbation of the disease was 31.4 years for the males and 32 years for the females. The average annual rate of attacks was 0.15/patient/year.

Our study demonstrated an increasing incidence of attacks during spring (29%) and summer (29.24%), with highest value in May (11%). The minimum incidence was in December (5.76%). This is maybe due to the increasing sunlight exposure and temperature or to seasonal viruses or allergens.

Most of the analysed patients were living in northern Greece and especially in Macedonia (83%). It was mentioned a significant difference in the prevalence of MS between the urban and rural population during the periods 1979-1992 and 1993-2008.

In the present study, the most frequent symptom during the onset of the disease was sensory disturbances followed by diplopia, optic neuritis and weakness.

The risk factors, reported by our patients, were: stress, surgical anaesthesia, CNS-acting drugs, viral infections, craniocerebral trauma and very high temperature.

We found five families with more than one MS member. The daughters of MS mothers have the greatest risk of demonstrating MS.

Further research is required to analyse the underlying mechanisms of MS.

P13. Increase of IL-6 levels is related with depressive phenomena in the acute phase of Multiple Sclerosis

Koutsouraki Euphrosyni1,2, Hatzifilippou Eleni2, Michmizos Dimitrios2, Cotsavasiloglou Christos1, Costa Vassiliki1,2, Baloyannis Stavros1,2
(1) 1st Department of Neurology, AHEPA Hospital, 54621
School of Medicine, Aristotelian University, Thessaloniki, Greece
(2) Neuroimmunological Laboratory of the 1st Department of Neurology, AHEPA Hospital, 54621, School of Medicine, Aristotelian University, Thessaloniki, Greece

ABSTRACT
There are still many doubts regarding the specific biological connection between multiple sclerosis (MS) and depression, as well as the association between depression and MS exacerbations. The relation between depression and MS remains an issue of debate.
Is depression a reactive condition to a chronic illness or yet another symptom of that illness?
Is there a biological connection between depression and MS remissions?
Is there a correlation between depression and the illness' progress?

It has been reported that depression is associated with immunological processes; however, we do not know definitely yet whether they are the cause or the consequence of depression. An increasing body of evidence clearly indicates that mediators of the immune mechanisms, such as cytokines and chemotactic factors, are of great importance in its pathomechanism. For the last decades the subject of cytokines became a central topic within multiple sclerosis research. Growing evidence suggests that these molecules play an essential role in the pathophysiology of MS, both by regulating aberrant autoimmune responses and by mediating myelin damage.

The investigation of the relationship between specific and widely used immunological markers and the acute phase of MS was the topic of this research. We attempted to indicate the possible connection between interleukin-6, acute phase of multiple sclerosis (MS) and depression.

We determined and statistically evaluated the levels of interleukin 6 (IL-6) and its soluble receptor (IL-6R) in the serum of 28 patients suffered mainly from the relapsing/remitting type of multiple sclerosis, during the acute phase of the disease, 14 MS patients in remission and 20 healthy individuals. Additionally we examined the presence of depression among the patients of the three groups and we attempted to correlate depression with the phase of MS and the levels of IL-6 and IL-6R.

The results of our study support that depression is not only very common during clinical exacerbations of MS, but also that the levels of IL-6 increase during the acute phase of the disease especially when depression is detected. Moreover, it seems to be a measurable association between the increased IL-6 and depression during the exacerbation of multiple sclerosis.

P14. Low bone density in women with Multiple Sclerosis

Papakonstantinou Stylianos[1], Sioka Xrysa[2], Pelidou Sygkliti-Henrietta[1], Fotopoulos Andreas[2], Tsouli Sofia[1], Georgiou Athanasia[2], Kalef-Ezra John[3], Kyritsis Athanasios[1].
1.Neurology Clinic, 2.Nuclear Medicine Laboratory and 3. Medicine Physics Laboratory, University Hospital of Ioannina

ABSTRACT

Purpose: to determine the possibility of low bone density in women with multiple sclerosis.
Materials and methods: the Bone Mineral Density (BMD) was measured by means of DXA in the lumbar spine and the left femoral bone in 41 women with multiple sclerosis (mean age 41.9 years). We also measured DXA in 45 healthy women (mean age 42 years), with no history of intake of substances that affect bone density, as control group. Subjects with t-score >= -1.0 were considered as normal, whereas subjects with t-score<1.0 were considered as abnormal, according to the lowest of two separated measurements of bone density. In addition, a whole-body DXA scan was performed in 21 of the women with multiple sclerosis and 22 of the women of the control group.
Results: 61% (25/41) of the patients with multiple sclerosis had low bone mineral density in the lumbar spine and/or the left femoral bone, against 38% (17/45) in the control group (p=0.036). No similar findings were recorded in the whole-body bone mineral density, where 80 % of the mass is solid bone tissue.
Conclusions: Subjects with multiple sclerosis have frequently low bone mineral density in areas with high percentage of spongy bone tissue. In women with multiple sclerosis, appropriate checking and medical intervention in certain cases is necessary, in order to decrease the possibility of bone fractures.

P15. HLA associations with multiple sclerosis in Greece

Ioanna Kouri, [1] Stylianos Papakonstantinou , [1,2] Vaios Mpempes , [1] Haris S. Vasiliadis, [3] Athanasios P. Kyritsis, [1,2] Sygkliti-Henrietta Pelidou [1,2]
1. Neurosurgery Institute, Medical School, University of Ioannina, Ioannina, Greece.
2. Department of Neurology, Ioannina University Hospital, Ioannina, Greece.
3. Orthopaedic Sports Medicine Center, Department of Orthopaedics, University of Ioannina, Ioannina, Greece.

Abstract

Background: Multiple sclerosis (MS) is a demyelinating inflammatory disease of the central nervous system originated by a complex interplay of environmental and genetic factors. The association of MS with the human leukocyte antigen (HLA) class II alleles was investigated in MS patients in northwest Greece, in the geographical region of Epirus.
Objective: Our aim was to estimate the prevalence of the HLA-DRB1*1501, HLA-DQB1*0602 and HLA-DQA1*0102 alleles, consisting the most common susceptibility haplotype in North European and North American Caucasians.
Methods: We studied 126 MS patients and 93 age and sex matched healthy controls. HLA typing was performed by a polymerase chain reaction (PCR) amplification with sequence-specific primers (PCR-SSP) method.

Results: We found that HLA-DRB1*1501, HLA-DQB1*0602 and HLA-DQA1*0102 alleles were significantly more frequent among patients (34% versus 11%, p = 0.00015; 69% versus 51%, p = 0.01; 76% versus 55%, p = 0.002, respectively). HLA-DRB1*1501, HLA-DQB1*0602, HLA-DQA1*0102 haplotype was significantly more common among patients (p = 0.00067). HLA-DRB1*1501 and HLA-DQB1*0602 alleles were more frequently detected in patients with initial symptoms from the brainstem or the cerebellum (p = 0.024). No significant correlation was observed among these alleles with sex, disease clinical course, or age at onset.
Conclusion: This is the first study to investigate genetic susceptibility to MS in Greece. Our results are in line with previous reports in North European and North American patients.

P16. Antibodies against GM1, GD1b and GQ1b in multiple sclerosis patients

E.S. Koutsouraki [1,2], E. Hatzifilippou [1], T. Banaki [1], V.G. Costa [1,2], S.J. Baloyannis [1,2]
1st Department of Neurology, Aristotelian University, AHEPA Hospital,Thessaloniki, Greece
[1]Neuroimmunological Laboratory, Out-patient clinic for MS patients

ABSTRACT
Gangliosides are a family of sialic-acid containing glycosphingolipids and they are highly concentrated in nervous tissues.
The aim of the present study was to correlate the titres of anti-GM1, anti-GD1b and anti-GQ1b antibodies with the type of MS, the duration of the disease, the disability and the therapeutic approach.
We examined the sera of 48 definite MS (multiple sclerosis) patients, according to I. McDonald et al. diagnostic criteria (33 females and 15 males), at a mean age of 38.0 years (±9.5) and 40 healthy individuals at a mean age of 39.0 years (±12.7) for high titres of anti-GM1, anti-GD1b and anti-GQ1b IgM, by the use of commercial available ELISA kits for anti-GM1 IgM.

CONCLUSION

❖ Increased titres of anti-GM1 and anti-GD1b IgM have been identified during clinical exacerbation of the disease in MS patients who demonstrated relapsing/remitting or secondary progressive type of the disease.

❖ Anti-GQ1b IgM was detected only in 35% (n=17) of the MS patients.

❖ Most of the patients who treated with IFN-B showed elevated values of anti-GM1 and anti-GD1b IgM. However, patients who treated with methylprednizolone also revealed high concentrations of anti-GM1 IgM/ anti-GD1b IgM.

❖ No significant correlation between anti-GM1 / anti-GD1b and the grade of disability or the duration of the disease could be found.

❖ However from the present investigation we could correlate the duration of the multiple sclerosis and the disability scale. Patients with a duration of MS >10 years revealed the highest scores in the EDSS (5.0-7.0) (p=0.0001).

P17. THERAPEUTIC REHABILITATION OF A PATIENT WITH NMO-POSITIVE RELAPSING LONGITUDINAL MYELITIS: A CASE REPORT

A. Ploumis,[1] S-E. Pelidou,[2] D. Varvarousis,[1] A. Kyritsis,[2] A. Beris[1]
1.Department of Surgery, Division of Rehabilitation, University of Ioannina Medical School
2. Department of Neurology, University of Ioannina Medical School

Objective: The purpose of this study is to describe the rehabilitation process of a paraplegic patient with longitudinal myelitis.
Methods: This a retrospective chart review of a 39 year-old patient with longitudinal myelitis and positive neuromyelitis optica (NMO) antigen. The disease started after a febrile episode and presented as spastic paraplegia with neurogenic bowel and bladder. CSF analysis was evident of inflammation and MRI showed cord edema from cervical to lumbar area. Steroids were administered IV. Thereafter, the patient started on rehabilitation. Physical therapy included exercises with tilt table and body weight suspension treadmill walking. .
Results. The patient was competent of bowel and bladder sphinchters within one month. In two months following initiation of the disease, patient was able to bear full body weight and walk with aid. On the fourth month, the patient was an independent ambulator with muscle strength measuring 5/5 in lower extremities bilaterally. Four months after initial diagnosis the patient showed a relapse with a paroxysmal burning pain in both lower extremities, lasting for some minutes, coming 8-10 times per day for 7 days. At this time point we had positive serum NMO antibodies tested for a second time. A new spinal cord MRI showed intensification of signal at C2-C4 cord levels with gadolinium enhancement on T1W images and the patient was started on an immunosuppression session with steroids and azathioprine and symptoms were managed with pain neuromodulators. In 7 days the patient restored initial function as before the relapse of the disease.
Conclusions. The rehabilitation process of patients with longitudinal myelitis requires intense physical therapy program, close observation for relapses and immunosuppression for long time recessions.

P18. Scientific Research- A Review

Fereshteh Sedaghat[1,2], Aliasghar Rakhshani[3], Stavros J. Baloyannis[1]
[1]Aristotle University Of Thessaloniki,[2] Nuclear Medicine Research Center- Mashhad University of Medical Sciences- Iran, [3] Department of Research- YETOS Company- Thessaloniki- Greece

The scientific method of research is said to be the means by which researchers are able to make conclusive statements about their studies with a minimum of bias. A standard method of research is better to be used by researchers in order to minimize the influence of conflicts of interest and biased opinions. We will have a short review of how a research is deemed to be scientific.

P19. ISCHEMIC CEREBROVASCULAR ACCIDENT IN THE DISTRIBUTION OF ANTERIOR CHOROIDAL ARTERY: A CASE REPORT

Thomas J. Tegos, Athinodoros Valavanis, Christos Pitsalidis,
Alexandros Chatziapostolou, Angeliki Papadimitriou, Stavros I. Baloyannis.
A' Department of Neurology, AHEPA Hospital, Aristotelian University of Thessaloniki,Greece.

Anterior choroidal artery is a branch of the internal carotid artery.

Immediately following its origin, exactly distally to the posterior communicating artery, is directed posteriorly, enters the choroidal fissure to supply the choroidal plexus of the temporal horn of the ateral ventricle and the walls of the lateral ventricle. It supplies the optic tract, uncus, hippocampus, amygdala, part of the basal ganglia(tail of the caudate nucleus, medial part of the globus pallidus), posterior and inferior part of the thalamus, part of the internal capsule(posterior and retrolenticular part), and also part of the pyramidal tract. It ends in the vicinity of the interventricular foramen (of Monro).

CASE: A 30 years old lady admitted to our department following an episode of loss of consciousness of sudden onset. Following an intravenous administration of steroids (methylprednizolone), the patient regained consciousness but a paralysis (initially flaccid and later spastic) of her right upper and lower limbs was noticed. In the cranial nerve examination, a right lower VII and XII nerve paralysis were noticed. An immediate brain CT without contrast was performed, demonstrating widespread edema and no evidence of focal ischemic lesion or hemorrhage or any other lesion. The patient was administered aspirin, gastric protection and intravenous steroids on regular basis.

There was no history of hypertension, diabetes, hyperlipidemia, heart disease, migraine, miscarriages, deep vein thrombosis, peripheral arterial disease. Two pre-syncopal episodes were mentioned in the past. The patient was a heavy smoker. There was no relevant family history.

In the Hospital the following blood examinations were undertaken and were normal: Full Blood Count, Erythrocyte Sedimentation Rate, C Reactive Protein, serum transaminases (SGOT, SGPT), glucose, lipid profile, urea, creatinine, sodium, potassium, calcium, Creatine Phospo Kinase, International Normalised Ratio, activated Partial Thromboplastin Time, homocysteine, cortisol, T3, T4, Thyroid Stimulating Hormone, CD4/CD8.

Rheumatoid factor, Anti Nuclear Antibody, anti-double-stranded DNA Antibody, Anti Neutrophil Cytoplasmic Antibody, Extractable Nuclear Antibodies screen were negative. C3 and C4 were normal. Anti-cardiolipin Antibody, lupus anticoagulant Antibody were negative, protein C, protein S, anti-thrombin III, fibrinogen, D-dimers, Fibrin Degradation Products were normal. Coagulation factors were normal.

On the second day from her admission, a contrast brain CT was performed, demonstrating an improvement in terms of the edema, but still no focal lesion.

On the third day from her admission, a brain MRI was performed demonstrating an infarct in the posterior limb of the left internal capsule with no enhancement(an anterior choroidal artery blood supply territory). MR-arteriography was normal.

On the eight day from her admission, a new brain MRI was performed, demonstrating no alteration of the aforementioned infarction and no other new areas of infarction. MR-venography was normal.Triplex of the carotid and vertebral arteries demonstrated no atherosclerotic plaques or dissection. Transcranial Triplex was normal.

Transthoracic and transesophageal echocardiography and a 24 hours Holter monitoring of the cardiac rhythm were unremarkable.

On electroencephalography, recurrent episodes of theta and delta waves in the left temporoparietal area were noticed.

SPECT demonstrated a reduction of the regional Cerebral Blood Flow in
the left temporoparietal area and signs of crossed cerebellar diaschisis.

Visual fields, visual acuity, fundoscopy and tonometry were normal.

On the twenty-first day from her admission the patient discharged from the hospital having achieved an excellent recovery. The patient discharged on clopidogrel and gastric protection.

P20. Mixed gliomas with desmoplastic copmponent: An adult case report

Balogiannis J., Syrmos N., Stavrinou P., Magras J., Polyzoidis K.
1st Neurosurgical Department AUTH, AHEPA University Hospital
Thessaloniki, Macedonia ,Greece

Mixed CNS tumors glial and mesenchymal component present a diagnostic and therapeutic challenge. These bimorphic neoplasms includes a wide spectrum of lesions, such as the gliosarcoma, gliofibroma and desmoplastic infantile astrocytoma (DIA) /ganglioglioma. In the literature there is only a single report of a desmoplastic glioblastoma Although there are some well documented cases in the literature, the rarity of these neoplasms contributes to the confusion regarding the nomenclature, classification and, more important, the treatment of these ailments. We report a case of a tumor with profound desmoplasia intermingled with a full-blown morphology of a glioblastoma . This is a unique case of a desmoplastic glioblastoma in an adult patient. A 53-year-old male presented with a 30-day history of increasing headaches,mild left-sided weakness and bilateral papilledema. MRI studies revealed a brain tumor. At surgery, the dura was found to be infiltrated . Although there was no clear arachnoidal plane, the tumor could still be separated from the surrounding brain parenchyma with relative ease, giving off the impression of a malignant meningioma. Gross complete resection was accomplished. Pathological examination confirmed the biphasic pattern and the mass was identified as a desmoplastic glioblastoma. The patient was subsequently treated whole brain radiation and he was started on Temozolomide. Forty-two months later he presented with a recurrence for which he was reoperated. This time the pathological examination was that of a typical glioblastoma multiforme.

P21. **Treatment of intractable increased intracranial pressure with decompressive craniectomy**

Balogiannis J., Syrmos N., Arzoglou V., Paraskevopoulos D., Tsitlakidis A.
1st Neurosurgical Department AUTH, AHEPA University Hospital
Thessaloniki, Macedonia ,Greece

AIM- Aim of this study was to examine the effectiveness of decompressive craniectomy in clinical practice with a special attention to long-term functional outcome . **MATERIAL- METHODS**- During a three year time period , 24 patients (18 male-75 %- and 6 female- 25 %- , range 16-77 years ,mean age 49,5 years) who underwent decompressive craniectomy were analysed retrospectively. There were:- group 1 , 7 patients with intracerebral haemorrhage (ICH) (29,2 %) - group 2 , 17 patients with severe traumatic brain injury (TBI) (70,8%) .
RESULTS -11 decompressive craniectomy patients (45, 8 %) did not survive to hospital discharge or to immediate after rehabilitation discharge . At a mean of 2 year (range, 6-36 months) after decompressive craniectomy, 7 survivors (29, 2%) had a poor whereas 6 survivors (25 %) had a good functional outcome .
CONCLUSIONS: The major benefit of decompressive craniectomy was observed in patients with a reduction in intracranial pressure . We have to improve patients selection and to optimize the timing of this procedure to have a better functional outcome in our patients .

P22. Suicidal behavior in the Psychiatric Department of General Hospital.

Tananaki M, Kiloudis P, Xerras C, Karafyles G, Mavroudis I, Mavridou E, Stathakis I
Psychiatric Department of Papanikalaou General Hospital of Thessaloniki, Greece

Suicidality is a broad term that includes both suicidal ideation and behavior, both nonfatal and fatal suicide attempts, that occur during drug treatment. There has been much focus on treatment-emergent suicidality in recent years, and the question of how best to assess this type of event in future trials has been raised. Suicidal behaviors and completed suicides are very common in psychiatric patients. The present study is a retrospective analysis of the nosological background of the patients that showed suicidal behavior or suicidal ideation from 2002-2010 and hospitalized in the Psychiatric Department of Papanikolaou General Hospital. The vast majority of the patients were suffered from mood disorders and borderline personality disorder, while substance abuse disorder were present in 18%, schizophrenia in 15% and anxiety disorders in 13%.

P23. Sports and recreation activities for young children in ancient Greece
The young boxers of Akrotiri ,Santorini (Thera)

Nikolaos Ch.Syrmos, Argyrios Mylonas
Department of Anatomy, School of Sports Science,Aristotle University of Thessaloniki, Macedonia,Greece

The Minoan civilization evolved some 4,000 years ago. Surrounded by sea, and with a strong fleet the Minoan evolved into a powerful thalasocracy (maritime supremacy). The Minoan town had almost no walls (like in later time Sparta and Macedonia). The Minoan civilization reached its peak by the year 2,000 B.C., and it maintained cultural and commercial links with the Egyptians, Assyrians and Babylonians.
The renowned sport of ancient minoan civilization, open to both genders and subjecting all to the same standards, was bull leaping. This was a dangerous pastime, but harmless and humane to the athlete and the animal if performed with skill. A bull would be released to charge toward the jumper. Once it was in sufficient proximity, the performer would attach his hands to the bull's horns and vault onto the creature's back. Another common objective was to somersault from such a position to a state of standing on a spot of land directly behind the bull.
Boxing was also a favorite activity. The precise regulations are unknown, but this is perhaps a source of inspiration for later Greeks, who adapted the sport to the Olympic Games. This fresco of Young Boxers is from Santorini (Thera) (1550 BC) represents the oldest known portrayal of a boxing sport, complete with gloves .
The painting depicts two boys with their heads shaved but for two long locks dangling at the back and two shorter ones above the forehead. They each wear a belt and have a boxing glove on their right hands. Their large exaggerated eyes are common in the Aegean frescoes of this period. The boy to the left wears jewelry consisting of a necklace and two bracelets, one on the arm and the other around the ankle. The other boy is unadorned, probably in order to indicate his lower social status. This scene may depict the boys taking part in an initiation rite.
.Made with a hand painted, aged appearance on a wood surface with brilliant blue and brown fresco hues .This wall painting was found in one of the houses in the settlement of Akrotiri iwhich was destroyed by the sixteenth-century BC volcanic eruption there. The Greek Bronze Age settlement is on the Greek island of Santorini, associated with the Minoan civilization due to close similarities in artifact and fresco styles.Frescoes, pottery, furniture, advanced drainage systems and three-storey buildings have been discovered at Akrotiri, whose excavation was started in 1967 by Spyridon Marinatos.Dating from the Bronze Age, late Bronze Age, 16th c. B.C., the fresco adorned the south wall of a second-floor room in House B, where other frescoes were also uncovered.

P24. Microcephaly in ancient Greece -The Minoan Microcephalus of Zakros

Nikolaos Ch. Syrmos

Microcephaly is a neurodevelopmental disorder in which the circumference of the head is more than two standard deviations smaller than average for the person's age and sex. Microcephaly may be congenital or it may develop in the first few years of life. The disorder may stem from a wide variety of conditions that cause abnormal growth of the brain, or from syndromes associated with chromosomal abnormalities. The eterogenity of the condition poses problems for clinical evaluation as well biological, genetic and antropological analysis .
These are the figures (Fig. 1, Fig. 2) of a microcephalic skull that was found on Zakros, one of the major Minoan (Knossos, Faistos, Zakros, Malia) archaeological sites of Minoan Crete (Fig. 3, *Fig. 4*) .
The Minoan civilization, a Bronze Age civilization, arose on the island of Crete and flourished from approximately the 27th century BC to the 15th century BC. It was rediscovered at the beginning of the 20th century through the work of the British archaeologist Sir Arthur Evans. Will Durant referred to it as "*the first link in the European chain"* .
The cranium was excavated from a Minoan Period grave dated about 4.000 years ago and was preserved and studied first by Dr. A. Poulianos in 1975 and later by Dr. G. A. Lyras *in 2009* .
The skull is of a person about twenty years old. It was buried in a very small chest-shaped coffin, which suggests a short stature for the individual. The width of the skull is narrow in comparison with the length, the frontal bone retreats, and there is alveolar prognathism and a well-developed mental prominence ago .
According to experts this skull belongs to *Homo floresiensis.* Homo floresiensis has been attributed to a species of its own, a descendant of Homo erectus or another early hominid, a pathological form of Homo sapiens, or a dwarfed Homo sapiens .
This fact reflects probably a genetic diversion connected to long terms isolation and endogamy of Minoans. Thus neither Cretans, nor members of the Minoan dynasty were immigrants from elsewhere, but a result of local evolution .

P25.Η συνοδεία και το σχέδιο δράσης σαν ένας τρόπος παρέμβασης για την βελτίωση της ποιότητας ζωής των χρόνιων πασχόντων

Δεμίρης Μόσχος Ψυχολόγος Κοινωνιολόγος, Δ/της Εταιρείας Σπαστικών Βορείου Ελλάδος

Η σταδιακή εξέλιξη των χρονίων παθήσεων φέρνει σε δύσκολη θέση τους φροντιστές του περιστατικού οι οποίο πραγματικά δυσκολεύονται στην φροντίδα του ασθενούς μιας και πλέον αυτός χάνει την αίσθηση της πραγματικότητας. Σ' αυτό το ολιστικό μοντέλο αντιμετώπισης των χρονίων παθήσεων το κέντρο είναι πάντα ο ασθενής γιατί αυτός είναι που υποφέρει πραγματικά, όμως θα πρέπει να γίνει κατανοητό πως στις χρόνιες παθήσεις μιλάμε για εξελισσόμενες ασθένειες και τα αποτελέσματα είναι όλο και πιο επιβαρυντικά για τους ασθενείς. Για το λόγο αυτό είναι απαραίτητο ένα πρόγραμμα συνοδευτικής δράσης με συγκεκριμένο στόχο και πολλούς επιμέρους στόχους, βασισμένο στις ανάγκες ενός προσώπου, και μόνο αυτού. Μια από κοινού ενέργεια δράσης συνοδού και συνοδευόμενου, που πρόκειται να υλοποιηθεί, προκειμένου να ικανοποιήσει ένα αίτημα του συνοδευόμενου και παράλληλα να τον οδηγήσει σε μια μεγαλύτερη δυνατή αυτονομία και ανεξαρτησία. Για την δημιουργία αλλά και εξέλιξη ενός λειτουργικού σχεδίου δράσης χρειάζεται η συνεργασία όλων των εμπλεκομένων και ιδιαίτερα των γιατρών. Είναι απαραίτητη η επιμόρφωση όσων αφορά την συνοδεία και τις θεραπευτικές της ιδιότητες. Θα πρέπει να κοιτάξουμε ολιστικά την θεραπεία των χρονίων πασχόντων.

P26. Out of sight, out of mind: The role of the hidden object in a novel memory screening test

Sokratis G. Papageorgiou, Department of Neurology, Eginition Hospital
Alexandra Economou, Department of Psychology, University of Athens

Objectives: To present preliminary data on the performance of healthy and memory-impaired individuals on a novel memory screening test. To relate test performance to neuropsychological test performance.
Methods: *Participants*: A community-dwelling sample of 81 middle-aged and older controls and a group of 62 memory-impaired patients (29 mild AD, 3 AD, 7 other dementia & 23 MCI) spanning a wide range of years of education (0-21), matched for education. *Task*: The 5 objects test requires the recall of the locations of 5 everyday objects, 4 of which are placed on the 4 corners of two adjacent sheets of paper (A4), and the 5th is placed in the pocket of the examiner. The examiner first guides the examinee in placing the 5 objects and then hands each object to the examinee in a specific order, to be placed in the correct location. The task is repeated until perfect performance is achieved, up to 4 trials, with a delayed recall after 5 minutes. *Procedure*: Controls received a brief neuropsychological battery that included a Greek research version of the California Verbal Learning Test (CVLT). Patients received a battery of tests that included the modified Mini Mental State Examination (mMMSE), the 5-words memory test (Dubois, 2000) and the Clock Drawing Test.
Results: Immediate and delayed recall performance was at ceiling for the controls (trial 1 mean 4.57 ± .96, trials 1-4 mean 19.41 ± 1.48, delayed recall mean 4.98 ± .16) and was nearly identical for a subset of age- and education-matched controls (N = 34). Patients scored significantly lower on all 3 measures (trial 1 mean 2.95 ± 1.25, trials 1-4 mean 14.48 ± 4.95, delayed recall mean 3.81 ± 1.41) (all *p*s < .001). Trial 1 scores showed the largest effect size and the hidden object led to the highest percentage of incorrect placement (88%) for the patients, differentiating the responses of the 2 groups the most ($x^2 = 78.94$, $p < .001$). No relationship was found between the 5 objects test scores and the CVLT for the control group, due to near ceiling performance on the former test. Correlations of the 3 scores of the 5 objects test with the MMSE and mMMSE were significant for the patients (all *r*s > .41, *p*s < .001). Correlations of the 3 scores with age and years of education were nonsignificant for both groups.
Conclusion: The 5 objects test is a quick, easy to administer and sensitive to memory impairment screening test that can be used with persons of minimal education.

P27. Receptive Aphasia as an early symptom of CJD

Makrydakis G., Anyfandi E., Liapis C. Bonakis A., Routsis C., Spilioti E., Papageorgiou SG., Kalfakis N.
1st Department of Neurology, Eginition Hospital, National and Kapodistrian University of Athens, Greece.
Aphasic disorders may be among the common features of CJD, but their occurrence as an initial symptom has not yet been adequately assessed. Here we present two cases of sCJD initially presenting with receptive aphasia. Patient M.E., 78 years old, male, presented with rapidly progressive receptive aphasia, exhibiting further cognitive decline, myoclonus and EEG alterations after 2 months of initial symptom onset. Brain MRI demonstrated DWI cortical ribbon-like hyperintensities and respective pathological ADC maps, while CSF 14-3-3 protein levels were elevated. ^{99m}Tc HMPAO - SPECT further demonstrated reduced further signal intensity in frontal, parietal and occipital areas of both cerebral hemispheres. Patient X.M., 68 years old, presented initially with rapidly progressive receptive aphasia as the only clinical symptom for 6 months. Further progression with myoclonus and mild apraxia led to a comprehensive investigation which revealed sCJD, confirmed by brain MRI (bilateral parietooccipital cortical ribboning in DWI ssequences, pathologic ADC maps). These two cases suggest that in cases with rapidly progressive aphasia, CJD should be included in the differential diagnosis.

P28. A case of abortive Hashimoto encephalopathy exhibiting a selective memory deficit.

C. Koros, A. Economou, G. Mastorakos, A. Bonakis, N. Kalfakis, S. G. Papageorgiou
Cognitive Neurology-Extrapyramidal Disorders Unit, 1st University Department of Neurology, Eginition Hospital, Medical School, University of Athens, Greece
Endocrine Unit, Aretaieion Hospital, Medical School, University of Athens, Greece
Department of Psychology, University of Athens, Greece

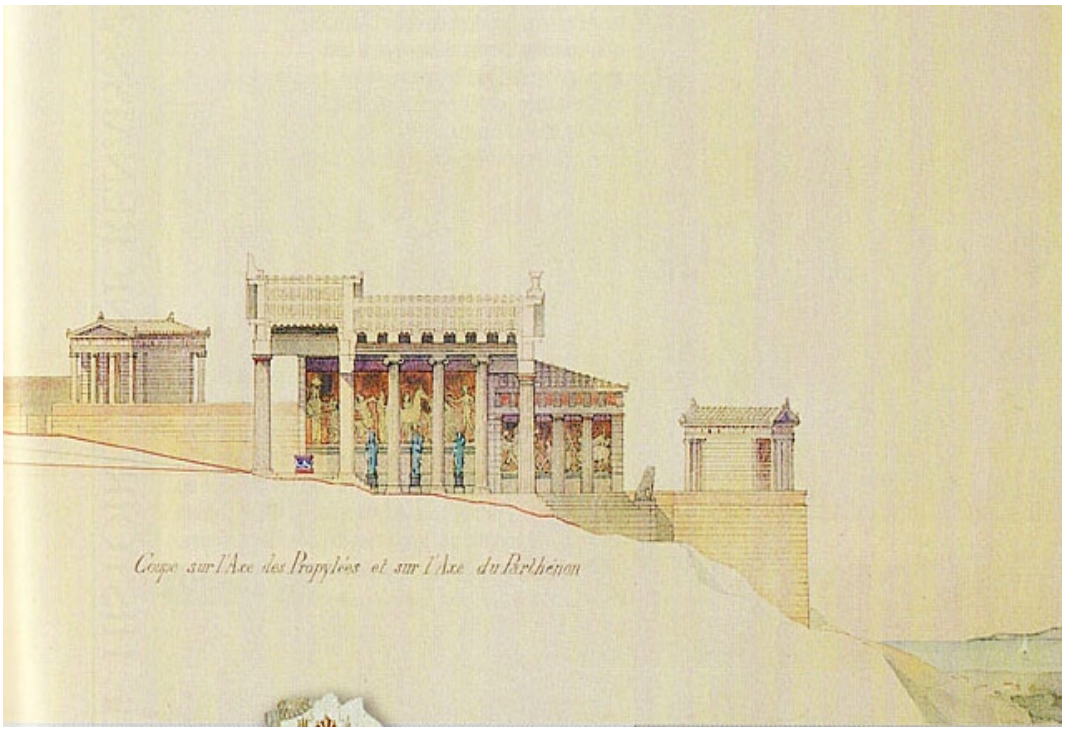
Objectives: We report a case of a long-standing selective memory deficit in a euthyroid (treated by L-thyroxin) 45 year-old female patient with Hashimoto thyroiditis. The patient complained of memory problems and deterioration of concentration skills, which started approximately two years before her present examination. It is of note that the patient didn't express any signs of depression, which had been proposed as a diagnosis by her endocrinologist.
Methods: Upon initial assessment, the patient underwent complete clinical examination, neuropsychological evaluation and routine blood investigations. Cerebrospinal fluid (CSF) examination, brain MRI and electroencephalogram (EEG) were also conducted. A complete follow-up evaluation took place after a four month interval.
Results: The physical examination was normal. The patient scored 30 points in the MiniMental State Examination. However, detailed neuropsychological testing showed a severe deficit in verbal memory (WMS-story recall). Blood and CSF investigations were normal except for serum anti-TPO which were elevated (452,4 reference range <9 IU/ml). Brain MRI was normal. EEG showed scarce intermittent bilateral multifocal theta waves. The patient was started on small doses of corticosteroid therapy and both her cognitive function and EEG features had returned to normal 4 months later.
Conclusion: Our case suggests that beyond the already known clinical picture of encephalopathy, HT can cause a chronic and selective memory deficit that can spare executive functions and attention. This clinical picture, which can be misdiagnosed as depression, can only be diagnosed with a detailed neuropsychological examination.

Author's Index

ACKNOWLEDGEMENTS

We wish to express our gratitude to the following companies who though their generosity, have helped to make this Congress possible.

Bayer Hellas
Genesis Pharma S.A.
Janssen-Cilag Pharmaceutical SACI
Novartis (Hellas) S.A.C.I.
Pfizer Hellas S.A


Coupe sur l'Axe des Propylées et sur l'Axe du Parthénon