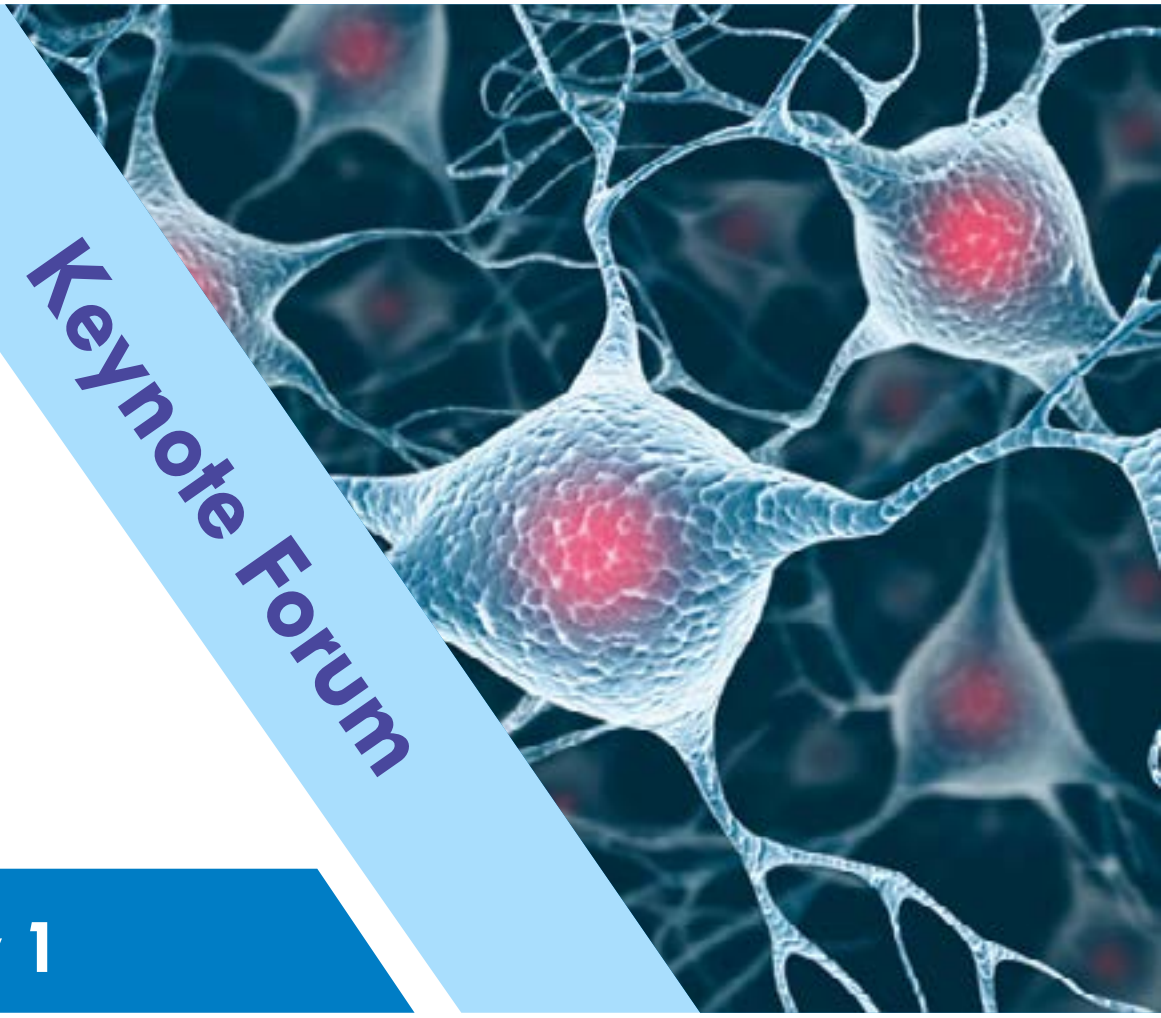
8th European Neurology Congress

September 21-23, 2016 Amsterdam, Netherlands



Keynote Forum

Day 1




Allal Boutajangout, J Neurol Neurophysiol 2016, 7:4 (Suppl)
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8th European Neurology Congress

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Allal Boutajangout

New York University School of Medicine, USA

Therapy and immunotherapy targeting Alzheimer's disease

Alzheimer's disease (AD) is an age-related progressive disorder characterized by the extracellular accumulation of amyloid β (Aβ) peptides as plaques and cerebral amyloid angiopathy (CAA), as well as intracellular neurofibrillary tangles (NFTs). AD is the most common cause of dementia globally. Care for patients with dementia accounts for ~1% of current global GDP, with this expected to rise substantially in the near future. No effective treatment is available to prevent or cure AD. Currently available treatments for AD provide largely symptomatic relief, with only minor effects on the course of the disease; hence, there is an urgent need for better therapeutic interventions. Aβ has become a major target for disease modifying treatments of AD. Unfortunately, the ongoing trials targeting amyloid Aβ failed in phase III trials. So far, the clinical benefits to the patients are limited and have no effect on tau related pathology. Previously, we reported for the first time, that active and passive immunotherapy targeting tau pathology reduces tau pathology and improves cognitive decline in two different NFT models. Recently, we developed a new monoclonal antibody against PHF-tau that reduces tau pathology and improves cognitive decline without inflammation. We have also explored the potential effects of hUCB-MSC on AD pathology. Our results suggest that use of these stem cells is associated with a reduction of amyloid burden, which correlates with improvements of cognitive function in a transgenic AD mouse model. These promising approaches using immunotherapy targeting tau or stem cells to reduce Aβ pathology in animal AD models provide critical data prior to potential clinical trials.

Biography

Allal Boutajangout graduated from Free University of Brussels (ULB-Erasme Hospital), School of Medicine (PhD in Neuropathology). He completed his Postdoctoral training at New York University School of Medicine. He is a Research Associate Professor of Neurology and Neuroscience & Physiology and Psychiatry. He is also the chief of Neurodegeneration and Drug Discovery Program within Center for Cognitive Neurology at NYU. He received prestigious award Margaret M Cahn for his outstanding research in the field of Alzheimer's and other awards from: Alzheimer association, NIH pilot grant, Toyama Company, Revalesio Company and co-investigator in 5 RO1 NIH grants. He has published more than 30 papers in reputed journals and serves as a reviewer for many scientific journals.

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Giovanni Antioco Putzu, J Neurol Neurophysiol 2016, 7:4 (Suppl)
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8th European Neurology Congress

September 21-23, 2016 Amsterdam, Netherlands

Giovanni Antioco Putzu

Casa di Cura Sant'Elena, Italy

Guillain-Barré Syndrome (GBS) and GBS-like Syndrome: Clinical, neuropathological and immunological correlations

GBS is an auto-immune life-threatening inflammatory polineuropathy that may produce severe functional disability. Vaccines, viral, fungine or bacterial infections may trigger the disease. On the basis of neuropathological and neurophysiological findings, GBS is classified in demyelinating and axonal forms. In both features, functional disability is directly correlated to axonal loss. Involvement of amyelinic axons is responsible for autonomic disturbances, which, along with bulbar spread of the disease, represent a potential cause of death in GBS. A consistent number of patients both in the early or recovery phases may complain of neuropathic pain that requires an adequate treatment. Immunological aspect of the disease, i.e auto-antibodies directed against GM1 and recently to contactin-associated protein 1 (Caspr) of the paranodal region of myelinated nerves, have already been investigated. We have demonstrated that TNF-alpha was immunolocalized in both myelinated and unmyelinated axons the sural nerve of GBS patients. We concluded that this substance may be directly responsible for axoal loss(G.A. Putzu et al, J. Neurol Sci, 2000). Interferon-gamma, which is a stimulator of IL28A was also easily detected in the sural nerve of GBS patients. The role of adhesion molecules like ICAM in the immune process of GBS will be also discussed. The therapeutic approach of GBS is aimed to avoid death in the acute phase (respiratory failure in Landry paralysis, cardiac rhythm anomalies in disautonomia). The efficacy of plasmapheris and intravenous immunoglobulins in the treatment of GBS is nowadays clearly demostrated. The next frontier is the theoretical possibility to use monoclonal antibodies (i.e, anti-INF-gamma) as a therapeutic tool in GBS. We also rewied the literature on GBS-like conditions that may clinically mimick GBS.

Biography

The Author is a Medical Doctor since 1992, with specialization in Paediatric Neurology. He achived his PhD in 1996. During PHD studies, He was a Research Fellow in Hammersmith Hospital of London, UK in 1992, then He moved in Marseille to work at INSERM (Genetics) and in Neurophathology. The Author has published more than 15 papers in the field of Neuromuscular Disorders.

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Peter Silburn, J Neurol Neurophysiol 2016, 7:4 (Suppl)
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8th European Neurology Congress

Peter Silburn

The University of Queensland, Australia

Biography

Peter Silburn is a Professor of Clinical Neuroscience at The University of Queensland, Director of the Asia-Pacific Centre for Neuromodulation, and a world expert in the treatment and research of Parkinson's disease, related neurodegenerative disorders and Deep Brain Stimulation (DBS). Professor Silburn's work in DBS is changing the lives of patients with a wide range of diseases and conditions for whom standard medical therapies have not been effective, including patients with Parkinson's disease, Dystonia, Tourette's syndrome, Essential Tremor, and Post-stroke disorders.

Professor Silburn graduated from The University of Queensland in 1988 and commenced training in neurology at The Princess Alexandra Hospital, completing his training at Oxford in The United Kingdom at the Radcliffe Infirmary. He was subsequently the Clinical Lecturer in Neurology at the University of Oxford.

He then went to the Karolinska Institute, Stockholm, as a Research Fellow in the Department of Molecular Medicine. He returned to Brisbane in July 1996, commenced private practice and established affiliations with The University of Queensland.

He became full Professor in Neurobiology in 2006 and Foundation Professor of Clinical Neuroscience at the UQ School of Medicine in 2007.

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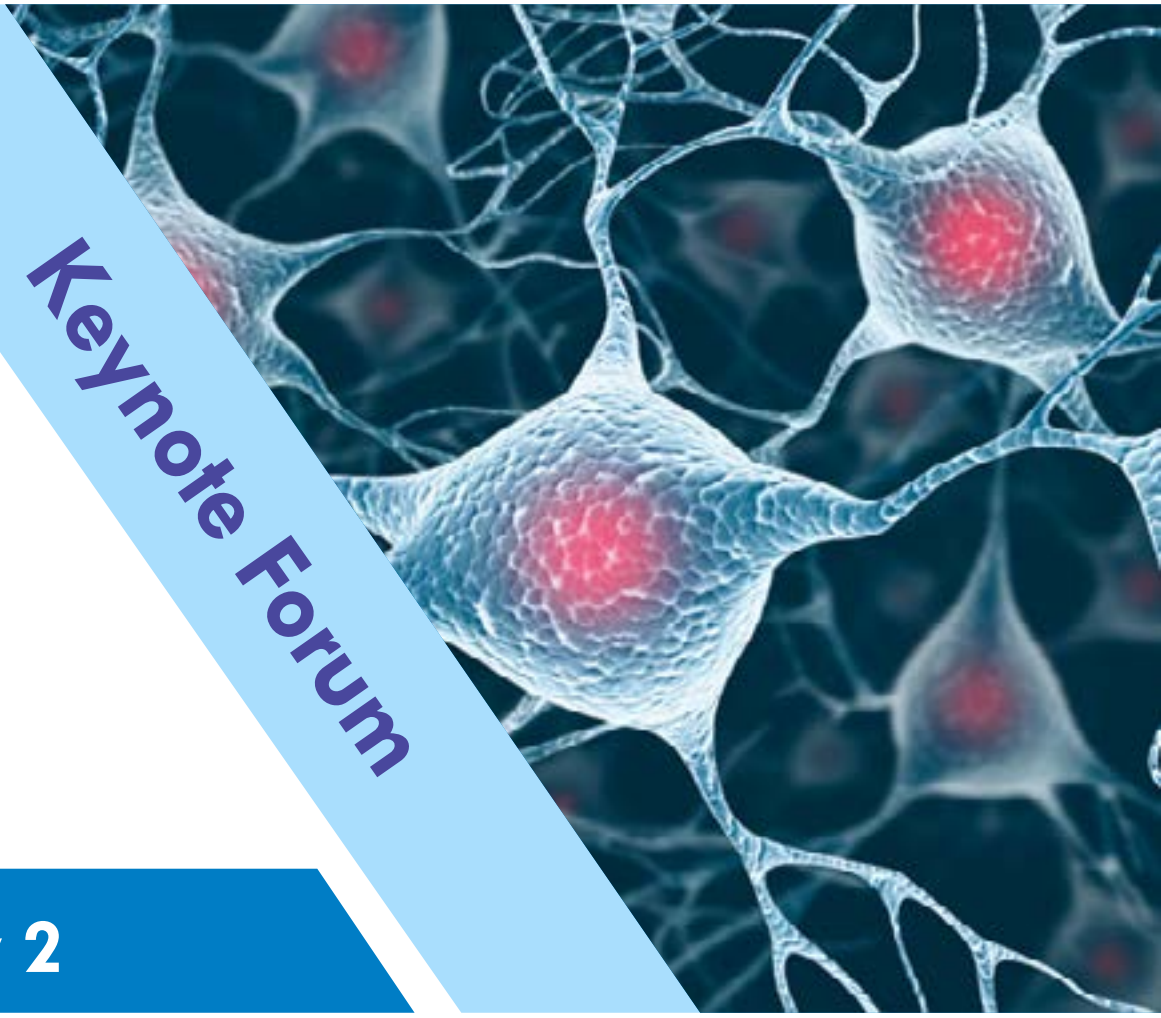

Keynote Forum

Day 2




Galina Mindlin, J Neurol Neurophysiol 2016, 7:4 (Suppl)
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8th European Neurology Congress

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Galina Mindlin

Mount Sinai, USA

Short-term effectiveness and acceptability of "Brain Music Therapy" (BMT), a self-guided neurofeedback intervention for anxious insomniacs

Objective: This uncontrolled pilot study assessed short-term effectiveness and acceptability of "brain music therapy" (BMT), a self-guided neurofeedback intervention for anxious insomniacs.

Methods: Following baseline assessment, volunteers (n=15) with clinically significant insomnia and anxiety underwent EEG. Slow and fast wave brain patterns were converted to piano music tacks and transferred to CD's. Participants were instructed to use their personalized CDs to facilitate sleep and anxiety reduction (relaxing track) or to stimulate focus and alertness (activating track) on a daily basis. Repeated measures of sleep (PIRS), anxiety (STAI), daytime functioning (DFT) and quality of life (QOL) were taken at weeks 0, 3 and 6.

Results: Participants were middle-aged (43.9/11.4), Caucasian (60.0%) females (66.7%) who were college educated (100%) and employed (93.4%). ANOVA showed significant changes on measures of sleep, anxiety and DFT (i.e., fewer negative effects); no changes were found for DTF (i.e., more positive effects) or for QOL. Intervention acceptance was high, with participants reporting easy use, helpfulness and willingness to refer friends with similar problems.

Conclusions: Results provide preliminary support for BMT as a treatment for anxious insomnia. The intervention is user friendly, while eliminating the need for potentially dangerous hypnotics and repeat visits to psychotherapists.

Biography

Galina Mindlin, MD is an Assistant Clinical Professor of Psychiatry at Icahn School of Medicine, Mount Sinai Health System and Clinical/Executive Director at Brain Music Treatment Center in New York City. She is board certified in Psychiatry/Neurology and holds a PhD in Neuroscience. She co-authored the book *"Your Playlist Can Change Your Life"* (Sourcebooks, 2012). She is trained in psychodynamic psychotherapy at Columbia University, completed her training in DBT and is thought mindfulness by Thich Nhat Hanh. She is the founder of Brain Music Therapy (BMT), in the US. She collaborates with other neurofeedback providers across the United States and is in private practice in New York City.

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Mohammed ELSherif, J Neurol Neurophysiol 2016, 7:4 (Suppl)
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Mohammed ELSherif

Mansoura University School of Medicine, Egypt

Diagnostic and Prognostic Significance of Blood Biomarkers in Acute Ischemic Stroke

Background: The utilization of biomarker panels in acute ischemic cerebral stroke (AICS) could enhance the proper diagnosis that facilitate the identification of the cause of the cerebral stroke which is essential for rationally manage and avoid stroke recurrence. Objectives: To inspect the vulnerable associations among a panel of blood biomarkers {D-dimer (DD), angiopoietin-1 (ANGPT1), S100 calcium-binding protein B (S-100b), and brain natriuretic peptide (BNP)} and AICS patients. Patients and Methods: This is a prospective research performed on patients with AICS who admitted at Saudi German Hospital-KSA in corporation with the neurology department Mansoura faculty of medicine - Egypt during one and half years' duration. Demographics of the patients, fatality as well as the clinic and a panel of blood biomarkers serum levels were gathered. The clinical scales {National institutes of Health Stroke Scale (NIHSS) scoring for severity on admission, and Modified Rankin Scale (mRS) for outcome after 3 months were tested for all AICS patients. Results: An overall of 150 patients with AICS was investigated, with a mean age of 62±14 years with males 52%. The AICS cases were set side by side to age and sex matched thirty healthy. controls (HC) demonstrating that the patients were more likely to have significantly hypertension, and atrial fibrillation (71.3%, 20%, P< 0.05 respectively). The mortality after 3 months was 11% (15 cases). Regarding stroke severity NIHSS score mean was 11.6±6. The serum levels for a panel of blood biomarker (DD, S100b, and BNP) are significantly higher while for Angpt1 is significantly lower with AICS in comparison to HC. Multivariate predictors of patients with an unfavorable functional outcome, DD, S-100b, and BNP levels were significantly higher compared with the levels in patients with a favorable outcome. On the contrary, the level of Angpt1 is significantly decreased in patients with an unfavorable functional outcome. The stroke severity (NIHSS score) correlated significantly with the outcome (mRS) as less severe cases showed more favorable outcome. The clinical variables that showed significant correlation were age, diabetic, and atrial fibrillation. Conclusion: Our findings highlighted that blood biomarkers can be accustomed as a valuable tool to investigate AICS and to anticipate initial neurological outcome that would assist in determining patients at risk of unfavorable outcome offering alert to launch therapies to avert aggravating of the patient's status.

Biography

Mohamed ELSherif has completed his MD and PhD at the age of 38 years from Mansoura University School of Medicine, Egypt. He is the coordinator of postgraduate and undergraduate medical students. He has published more than 17 papers in reputed journals and has been serving as a reviewer member of many Neurology journals. Prizes first Best Master Thesis Mansoura University 2007, second Junior Travelling Fellowships from the World Federation Of Neurology 22/4/2009 to attend the 13th EFNS in Florence-Italy to present the poster of my MD thesis.

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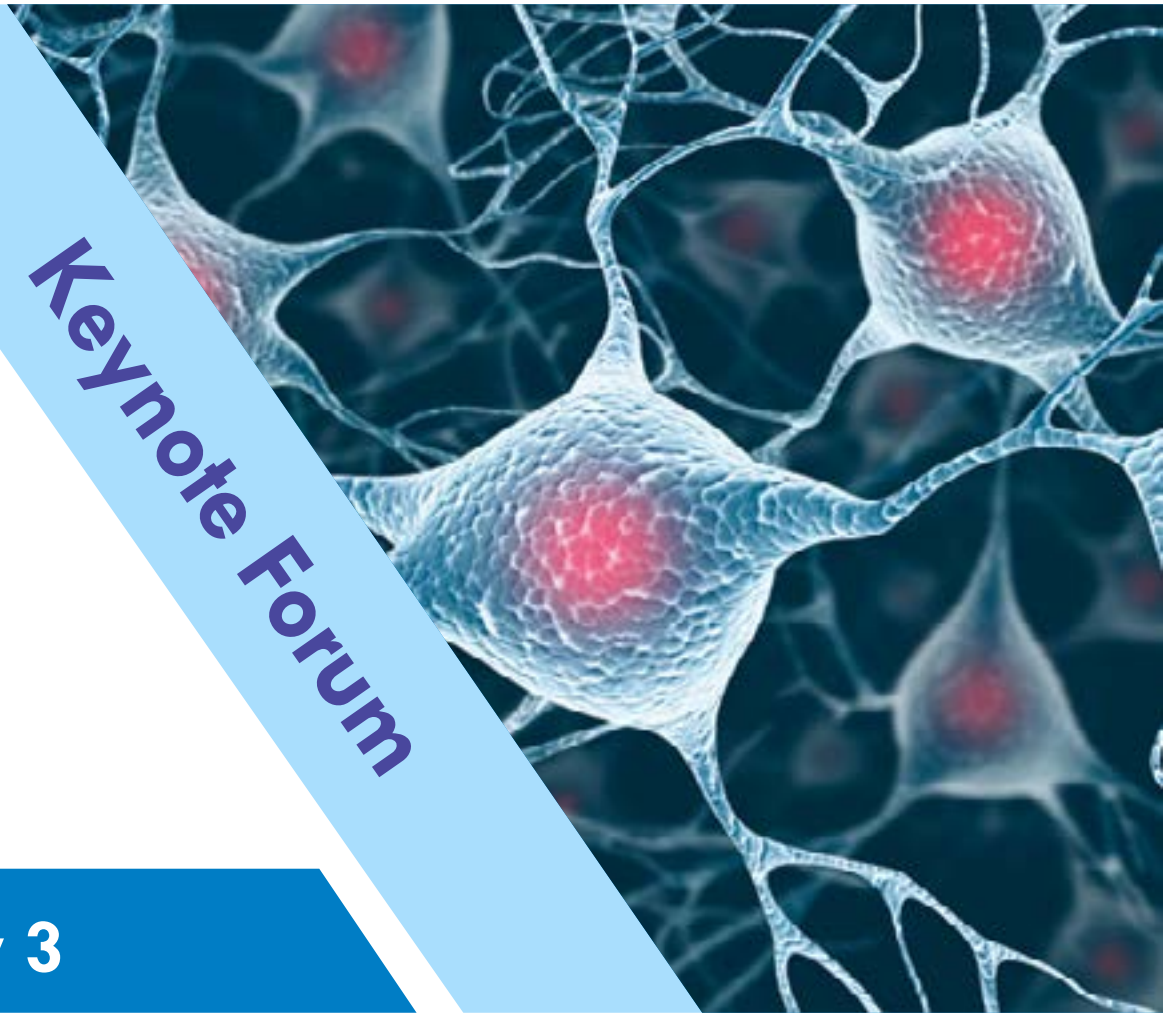

Keynote Forum

Day 3




Salvatore Polizzi, J Neurol Neurophysiol 2016, 7:4 (Suppl)
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Salvatore Polizzi

Medicina Occupazionale e Ambientale, Italy

Neurotoxic Effects of Aluminium and Alzheimer's Disease: Single Photon Emission Computed Tomography (SPECT) imaging among retired foundry workers with mild cognitive impairment (MCI).

Introduction: Aluminium is a well known neurotoxic metal and in a previous study (Neurotoxicology 2002; 23:761-774) we found a probable relationship between inhalation of aluminium dusts and MCI. The present study examined SPECT in a small subgroup that accepted to perform the test.

Methods: In a cross sectional study we enrolled 29 subjects: 20 professionally exposed to Aluminium, 9 control without any known occupational exposure to the toxic metal.

We determined SPECT imaging, serum levels of Aluminium (Al-s) and Iron, blood levels of Manganese and different neurocognitive tests: Mini Mental State Examination score (MMSE-score), the time to execute the test (MMSE-Time), Clock Drawing test (CDT) and auditory event related evoked potential ERP-P300 (P300).

Results: Al-s levels in the controls were lower than 10 µg/L. (8,6 ± 1,5 µg/L) and none showed SPECT hypoperfusion.

In foundry workers, Al-s levels were significantly higher ($p < 0,02$) (12,9 ± 1,5 µg/L) and SPECT imaging was normal in two subjects (10%), while 18 (90%) showed some degree of cerebral hypoperfusion: 14 (70%) revealed hypoperfusion in the temporo mesial hippocampus, para hippocampal region, and frontal cortex and 4 (20%) showed hypoperfusion in the cerebral cortex.

Conclusions: Even if the small size of the studied population imposes prudence in the interpretation of the results, SPECT hypoperfusion seems to be compatible with aluminium exposure; this could be controlled implementing lifestyle/diet (physical activity, curcuma, silica enriched water) to slow the brain ageing, to reduce body burden and to chelate the metal.

Biography

Salvatore Polizzi has completed his School of Medicine in 1981, at the age of 24 years, from Turin University School of Medicine. He completed his Occupational Medicine residency program from Turin University School of Medicine in 1988. He is the director Occupational and Environmental Medicine since 1984 and of Oncological Screening Program since 2000. He has published more than 60 papers in reputed journals both national and international.

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Notes:

Klaus Fritz, J Neurol Neurophysiol 2016, 7:4 (Suppl)
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8th European Neurology Congress

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David Rowell

Queensland Brain Institute, Australia

An economic evaluation of deep brain stimulation for patients with Tourette's syndrome: An initial exploration

Background: Tourette's syndrome (TS) is a neuropsychiatric movement disorder. Symptoms of severe TS include involuntary tics, vocalizations and coprolalia, which can progress to affect adversely health related quality of life. Co-morbidities include attention-deficit/hyperactivity disorder (ADHD), obsessive-compulsive disorder (OCD) and affective disorders. Typically, treatment may involve pharmacotherapy and supportive counseling. For a small number of patients, deep brain stimulation (DBS) is now being used to treat intractable TS. The clinical outcomes have generally been positive. To date, no economic evaluation of treating TS with DBS has been published. A well-designed economic evaluation has become a pivotal ingredient to ensure that the necessary resources are directed towards the healthcare services, which offer the best patient outcomes. The aim of this research is to present an initial exploration of an economic evaluation of DBS to treat severe TS.

Methods: We conduct a cost utility analysis (CUA), which compares the direct medical costs reported as $US and outcomes reported as quality-adjusted life years (QALYs) of DBS with best medical treatment (BMT). Our sample consists of 17 patients who received DBS for severe TS at St Andrews War Memorial Hospital, Brisbane, Australia from September 2008 to February 2012. Clinical indices for (i) tic severity (Yale Global Tic Severity Score) and (ii) depression (Hamilton Depression rating Scale) and (iii) age were collected pre and post DBS. These clinical data were converted QALYs using standardized coefficients derived from a multivariate regression published by Müller-Vahl et al (2010) for a sample 200 German outpatients (R 2 = 54%). The direct costs for DBS included hardware, surgical implantation, inpatient stay, neuro-stimulator programming and adverse events. For BMT direct costs included estimates for rehabilitation, inpatient stay, outpatient treatment, pharmaceuticals and ancillary treatments. All costs were reported in $US2016. TreeAge® software was used to estimate an Incremental Cost Effectiveness Ratio (ICER) using a Markov model, with a 10-year time horizon and 3.5% discount rate.

Results: The direct costs of DBS and BMT were estimated to be $USD 124,400 and $USD 34,180, respectively. DBS was estimated to increase health utility. The ICER was estimated to be lower than the $USD 50,000 per QALY threshold used by the Federal Drug Administration (FDA).

Conclusions: Our initial exploration suggests DBS is a cost-effective treatment for patients with severe TS. However, our economic evaluation contains several limitations. Firstly, indirect costs were not included. Secondly, health utilities pre and post DBS were imputed from clinical data rather than measured directly. Thirdly, long-term costs and benefits are uncertain; an average age of 28 years at implant implies a further 50 years of life post DBS. The ICER was sensitive to estimates of adverse events. Finally, our results were derived from a small sample. Future research will administer a survey of healthcare costs and QALYs to an international database of TS patients treated with DBS maintained by the University of Florida, with the aim of developing a more robust economic evaluation.

Biography

David Rowell is Post-doctoral Research Fellow in Asia-Pacific Centre for Neuro-modulation at Queensland Brain Institute. He has published more than 20 papers in reputed journals.

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