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October 2019 | Volume 3
Journal of Neurology and Clinical Neuroscience

Souvenir

3rd World Congress on

Neurology and Therapeutics

October 07-08, 2019 | Madrid, Spain



Hosting Organization: Pulsus

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Scientific Program

Scientific Program

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Registrations
09:00-09:30

Opening Ceremony
09:30-10:00

Day 1: October 07, 2019

Conference Hall: AYALA

Keynote Forum

10:00 - 10:45

Title: AmyP53: The first molecule able to block the very first step of the diseases, Alzheimer's and Parkinson's, non-toxic, easy to administer, easy to produce

Jacques Fantini | Aix-Marseille University | France
Nouara Yahi | Aix-Marseille University | France

Group Photo

Networking & Refreshments 10:45 - 11:05 @Lobby area

11:05 - 11:50

Title: Targeting of TYR682 residue of Amyloid Precursor Protein (APP) in Alzheimer's Disease patients for the development of new diagnostic and therapeutic tools

Carmela Matrone | University of Aarhus | Denmark

11:50 - 12:35

Title: Aspects on patients with chronic low back pain in Rehabilitation practice

Zaiga Kalnberza-Ribule | National Rehabilitation Center Vaivari | Latvia

Special Session

12:35 - 13:05

Title: Content validation of the international classification of functioning, disability and health core set in Latvian Stroke patients

Zaiga Kalnberza-Ribule | National Rehabilitation Center Vaivari | Latvia
Dana Svike | National Rehabilitation Center Vaivari | Latvia

Panel Discussion

Lunch Break 13:05 - 14:05 @Laventae

Workshop

14:05 - 15:00

Title: Insights on Amypore

Philippe Crespo | Amypore | France
Driss Fantini | Amypore | France

15:00 - 15:30	**Title: Saliva biomarkers for use in Alzheimer´s Disease** **Gorka Orive** | Geroa Diagnostics | Spain

Sessions: Neurology | Neuroscience | Neurological Disorders | Neurotherapeutics

Session Chair: **Zaiga Kalnberza-Ribule** | National Rehabilitation Center Vaivari | Latvia

Session Introduction 15:30 - 15:40

15:40 - 16:10	**Title: Novelo-Merida: The first Mexican Physiotherapy method based in Neuroscience** **Novelo Pérez Fernando** | Neurodevelopment center | Mexico

Networking & Refreshments 16:10 - 16:30 @Lobby area

16:30 - 17:00	**Title: The diagnostic yield of Hypotonia investigations: A 10-year single center study** **Mohammed Hassan AlBanji** | King Abdulaziz University | Saudi Arabia
17:00 - 17:30	**Title: The mediator effect of cognition on the relationship between Brain lesion location and Dysphagia in patients with Stroke: Applying a structural equation model** **Shiva Ebrahimian Dehaghani** | Shiraz University of Medical Sciences | Iran
17:30 - 18:00	**Title: A case report of Myasthenia Gravis treated with Plasmapharesis** **Theresia Christin** | Sriwijaya University | Indonesia

Panel Discussion

Poster Presentation 18:00 - 19:00

PP01	**Title: Clinical and diagnostic values of the determination of CSF specific proteins in Alzheimer's Disease patients** **Alessandro Rabbito | Medical University of Białystok | Poland**
PP02	**Title: Hearing screening of infants: BERA values, guidelines, and comparative programs** **Waleed B Shuaib | Kuwait University | Saudi Arabia**
PP03	**Title: Endocannabinoidome Tone in Multiple Sclerosis patients** **Alessandro Rabbito | Medical University of Białystok | Poland**
PP04	**Title: Brainstem Auditory Evoked Potentials (BAEP) during development and aging** **Eesa W Alshuaib | National University of Ireland Galway |Ireland**
PP05	**Title: A case report of Myasthenia Gravis treated with Plasmapharesis** **Theresia Christin | Sriwijaya University | Indonesia**

Panel Discussion

Day 02: October 08, 2019

B2B Meetings & Networking Sessions

Certification & Prize Distribution

Closing Ceremony

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Keynote Forum

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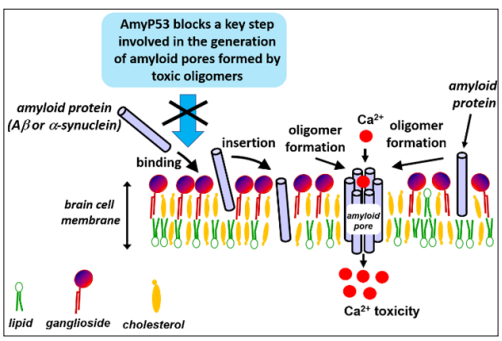
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Jacques Fantini[1]
Nouara Yahi[1], *Coralie Di Scala*[1], *Henri Chahinian*[2], *Driss Fantini*[2]
[1]*Aix-Marseille University, France,* [2]*Amypore, France*

AmyP53: The first molecule able to block the very first step of the diseases, Alzheimer's and Parkinson's, non-toxic, easy to administer, easy to produce

Large aggregates of amyloid proteins have long been considered as the main culprits in Alzheimer's disease (AD) and Parkinson's disease (PD). However, all therapeutic approaches of AD based on the clearing of amyloid plaques have failed and/or been abandoned. Hence, the old paradigm linking amyloid plaques and neurodegenerative diseases is no longer valid. Nowadays, low-molecular weight aggregates called oligomers are known to be the primary toxic agents responsible for neuronal dysfunction causing AD and PD. It is also well established that membrane lipids are critical members of the amyloid interactome, inducing the formation of neurotoxic oligomers of Alzheimer-β amyloid peptide (Aβ) and α-synuclein. These oligomers, referred to as amyloid pores, are responsible for a calcium overdose leading to ROS overproduction and Tau hyperphosphorylation, which in turn trigger important functional perturbations in brain cells and finally their degeneration and death. Our team deciphered the biological code controlling the formation of amyloid pores in human brain cells. We identified gangliosides as the primary target of amyloid proteins on neurons and astrocytes and determined which part of these proteins is involved in ganglioside recognition. With the help of Artificial Intelligence, we then designed a chimeric peptide based on the ganglioside-binding domains of Aβ and α-synuclein, and modified it to improve ganglioside recognition. This chimeric peptide, now called AmyP53, prevents the formation of both Aβ and α-synuclein oligomers at nanomolar concentrations. AmyP53 is highly soluble in water, stable, nontoxic, and it crosses the blood-brain barrier. It is also active against mutants of Aβ and α-synuclein (responsible for familial forms of the diseases). It can be produced at large scale and low cost (100 times less expensive than immunotherapies) and is planned to be administered intranasally. Overall, AmyP53 already appears as the most promising solution for Alzheimer's and Parkinson's diseases.



Biography

Jacques Fantini from Aix-Marseille University and Institut Universitaire de France has over 30 years of teaching and research experience in biochemistry and neurochemistry areas. His current research is focused on the molecular organization of the synapse in physiological and pathological conditions. Jacques Fantini and Nouara Yahi deciphered the ganglioside code used by amyloid proteins to bind to the surface of brain cells and then the molecular mechanisms of amyloid pore formation by small oligomers of Alzheimer's beta-amyloid peptide and alpha-synuclein. These discoveries have allowed the design of a chimeric peptide (now called AmyP53 by AmyPore) with broad anti-amyloid pore activity, opening new avenues for innovative therapeutic strategies against Alzheimer's and Parkinson's diseases. Website: https://jfantini.jimdo.com/

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Carmela Matrone[1,2]

[1]University of Naples Federico II, Italy, [2]University of Aarhus, Denmark

Targeting of TYR682 residue of Amyloid Precursor Protein (APP) in Alzheimer's Disease patients for the development of new diagnostic and therapeutic tools

Alzheimer's disease (AD) is one of the main causes of disability in the aged population approaching pandemic proportions. So far, no neuroprotective therapy is available for AD which calls for mobilization of the research community to address this public health threat. Convincing evidence point on the increased production and the aberrant accumulation of Aβ peptides as leading cause of AD. To be processed and generate Aβ, the Amyloid precursor protein needs to be transported to the acidic neuronal compartments where the enzymes responsible for the APP cleavage are mostly active. The APP transport toward these compartments depends on the Tyr682 residue located on the 682YENPTY687 C-terminal motif of APP. Accordingly, we reported that the increased Tyr682 phosphorylation causes the aberrant accumulation of APP in acidic neuronal compartments thus leading to Aβ production. Data from neural stem cells, fibroblasts and recently from blood cells indicate that APP Tyr682 phosphorylation levels are increased in AD patients during the early stages of the pathology. Such APP Tyr682 phosphorylation was triggered by Fyn Tyrosine kinase in AD neurons. As such, Fyn tyrosine kinase inhibitors, Saracatinib and Masitinib, were able to reduce APP Tyr682 phosphorylation levels in AD neurons and to prevent Aβ production. All together these findings suggest that APP Tyr682 residue can be targeted for a personalized therapy in AD patients in which increased levels of APP Tyr682 phosphorylation are detectable.

Biography

Carmela Matrone obtained her PhD in the field of Biomedical sciences at the Medical faculty University Federico II of Naples where she is currently employed as associate professor in Pharmacology and where she has established her independent group working on Alzheimer's disease and related diseases. She was trained as post doc at European Brain Research Institute in Rome working with prof RL Montalcini (Nobel prize for NGF discovery in) mostly acquiring competencies and skills in cell biology working in Alzheimer's disease. In 2012, she moved to the University of Health in Aarhus, Dept of Biomedicine, (DK) where she became associate professor in pharmacology in 2014 and where she is currently a guest lecturer. She is involved in extensive international collaborations with eminent scientists and she is member of several associations in the Neuroscience field. She has authored more than 50 scholarly publications. She has made approximately 50 research and professional presentations at international, national, regional conferences as speaker, moderator, as well as an organizer. She is an experienced peer reviewer for international scientific journals and as well as for international granting agencies in the Alzheimer's disease field.

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Zaiga Kalnbērza Ribule

Dana Svike, G Berzina

National Rehabilitation Centre Vaivari, Latvia

Aspects on patients with chronic low back pain in Rehabilitation practice

In chronic low back pain treatment individual rehabilitation plays important role. Individuals who suffer with chronic pain often reject the notion that pain can be psychological or social, and often seek medical reasons to explain their pain. In many cases chronic pain is not associated only with medical reasons. The biological and neurological mechanisms of acute pain have been identified, but within the last decades, there has been a shift in examining chronic pain from a psychological perspective.

Biopsychosocial approaches to chronic pain focus on the complex interaction among the biological, psychological and social factors that are inherent in our experiences. According of ICF Core sets for low back pain the rehabilitation can be individualized for each patient and it plays important role in goal attainment. ICF is the WHO framework for measuring health and disability at both individual and population levels. These categories can be taken into account when conducting comprehensive, multidisciplinary assessment. Research show that psychological factors such as emotional distress and dysfunctional pain coping play a role in the development and maintenance of chronic low back pain, and it has emotional and behavioral consequences that influence the development of problems and outcome of treatment, and these may have more influence on disability and quality of life.

Increasingly widespread acceptance of the biopsychosocial model, along with the relatively modest performance of monotherapies, has led to increased research into the effectiveness of multidisciplinary rehabilitation. Main goals of the rehabilitation programs are: to reduce impairment, pain reduction, to provide improvement of functional abilities in activity and participation domains, to help the patient to return to productive life, to adapt physiotherapy program for persons with low back pain. As chronic pain is described as multidimensional, there is necessary to pay attention not only to medical treatment of low back pain but to psychological factors influencing low back pain disorder as well as coping. In National Rehabilitation Center Vaivari we preformed study to examine stress coping among patients with low back pain in rehabilitation practice in Latvia.

Our results showed that almost all stress coping strategies scores for patients with low back pain in their first visit are higher than in patients with revisit to Physical Medicine and Rehabilitation doctor and there is statistically significant difference in the use of problem oriented stress coping. According to our data there were strong interrelationships of disability and Pain Catastrophization in all subscales. The research data showed significantly higher scores of Pain catastrophizing in patients with more severe disability. Achieved results highlight the importance of a multifactorial approach to pain management and the enormous significance of pain catastrophizing in patients with low back pain. It is critical to recognize these factors in both the acute and chronic pain patients in order to understand which aspects of their pain are a barrier to their recovery. The biopsychosocial approach view illness as the result of the dynamic interaction between psychologic and social factors and is widely accepted, but unfortunately not fully implemented. Therefore there is a necessity of the development of interdisciplinary pain management and rehabilitation approach programs for patients with low back pain.

Biography

Zaiga Kalnbērza Ribule, Vice President of Latvian Physical and Rehabilitation medicine Association, Member of European Union FRM doctors, Head of the department in National Rehabilitation Center Vaivari; department of General rehabilitation- Head of department. 2014 till present. Hospital of Kuldiga; department of Rehabilitation. Physical medicine and Rehabilitation 2013 til present (FRM doctor), Rīgas Austrumu klīniskā universitātes slimnīca (Riga university hospital). Physical and rehabilitation medicine physician. 2014 till 2019.

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Gorka Orive
Geroa Diagnostics, Spain

Saliva biomarkers for use in Alzheimer's Disease

Current therapeutic strategies in Alzheimer's disease (AD) are focus on the elimination of Aβ plaques. However, clinical benefits are poor as they are applied when the disease is already advanced. To succeed, all these approaches and future emerging therapies should be applied at a very early stage of the disease (before clinical symptoms appear). We have discovered and preliminary tested in a significant number of patients an accurate and non-invasive tool to identify candidates that suffer from mild cognitive impairment (MCI) and AD. This finding may revolutionize the fields of diagnostics, pharmaceutical development and decision making for patient care. We were able to detect a specific molecule (biomarker) in saliva that may help to identify those patients with MCI and/or AD from healthy status. I will summarize all our recent discoveries in the discovery and validation of the saliva biomarker and will compare them with other recently reported biomarkers such as blood biomarkers. Our results may represent a giant step forward for developing more accessible, less invasive, and easier predictive and diagnostic methods, thus making sense to the current efforts being conducted by the pharmaceutical industry. Additionally, it may help to understand patterns of cognitive decline and to discover new therapeutic targets.

Biography

Gorka Orive, founder of Geroa Diagnostics, a biotechnological company whose mission is to improve the health, the life-quality and medical care of patients suffering from Alzheimer's disease (www.geroa.net). Full-time Associate Professor of Biopharmaceutics, Pharmacokinetics and Pharmaceutical Technology, University of the Basque Country, Spain. Scientific Advisor of the Regenerative Medicine Laboratory at Biotechnology Institute ImasD (BTI), Vitoria, Spain. Researcher at the Spanish Research Network CIBER-BBN. 200 international scientific publications, 35 book chapters, 3 books and 3 patents. H-index:34*. Received several scientific awards and member of different international scientific societies. Principal Researcher of 10 national scientific projects. He has also been able to lead a number of projects as a main investigator (Spanish National Industrial projects: RETOS, Basque Government projects: GAITEK, SAIOTEK). Overall, he has advised 8 PhD students.

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International classification of functioning, disability and health core set in Latvian Stroke patients

Zaiga Kalnberza Ribule, Dana Svike, G Berzina
National Rehabilitation Centre Vaivari, Latvia

Purpose of the study: Content validation of the International classification of functioning, disability and health core set in Latvian stroke patients.

Method: This is a qualitative study involving 18 participants (8 men and 10 women) with a first-time stroke who were interviewed 3 to 4 years after the stroke. The average age of patients – 48 to 85 years. The interviews were transcribed and textual content was analysed, trying to recognize the functioning disorders that patients face on a daily basis. Afterwards they were further linked to ICF core set for stroke patients based on generally accepted binding principles.

Results: A total of 547 meaningful units were selected. 515 units were linked to the ICF stroke set. 31 meaningful units, which are in the ICF classification but not in the stroke set together were mentioned in 16 interviews. 1 unit could not be classified in ICF. The results of the study shows that in Latvian stroke patients following stroke group components were approved – 'Body function' - 33 codes of 41 code, 'Body structure' - 2 codes of 5 codes, 'Activities and membership'- 34 codes of 51 code, 'Environmental factors' - 20 codes of 33 codes.

Conclusions: The study confirmed 89 from 130 codes or 68%. The ICF stroke set was mostly approved. The ICF stroke set is a good tool that can be used in everyday practice to assess the patient's functioning limitations.

Biography

Zaiga Kalnbērza Ribule, Vice President of Latvian Physical and Rehabilitation medicine Association, Member of European Union FRM doctors, Head of the department in National Rehabilitation Center Vaivari; department of General rehabilitation- Head of department. 2014 till present. Hospital of Kuldiga; department of Rehabilitation. Physical medicine and Rehabilitation 2013 til present (FRM doctor), Rīgas Austrumu klīniskā universitātes slimnīca (Riga university hospital). Physical and rehabilitation medicine physician. 2014 till 2019.

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Workshop

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Insights on Amypore

Philippe Crespo and **Driss Fantini**
Amypore, France

Introduction: AmyPore, a biotech on its way to cure Alzheimer's and Parkinson's diseases

Until now there has been no cure for Alzheimer's (AD) and Parkinson's (PD) diseases. Several approaches targeting secretases and immunotherapy against amyloid deposits have been tested in patients at various stages of the disease. Similarly, immunotherapies against tau have also been attempted, but all those preclinical and clinical studies have failed or have been inefficient.

Instead of being caused by amyloid plaques, AD and PD are in fact characterized by oligomeric formation of beta-amyloid and alpha-synuclein proteins, respectively. These small oligomers are toxic and membrane-permeable, allowing a massive entry of Ca2+ into brain cells and ultimately cell death. AmyPore designed small peptides capable to inhibit the oligomerization of beta-amyloid and alpha-synuclein proteins, showing strong evidence for a common mechanism of neurotoxicity in AD and PD. The lead candidate, AmyP53, is a first-in-class peptide that is:

1. Efficient, in tackling the very first disease step
2. Non-toxic:
   - by design
   - by dosing
   - by demonstration
3. 100 times less expensive than any immunotherapy
4. Quick scale-up, worldwide.

About AmyPore: After more than 15 years of disruptive research, our inventors, Prof. N. YAHI (92 scientific articles ; H-index: 42) and Pr. J. FANTINI (190 scientific articles ; H-index : 54) found and patented the most promising solution, for AD and PD. Their research has been supported and validated by two Nobel and one Lasker laureates. AmyPore has an exclusive 15y license to value this solution, now called "AmyP53". www.amypore.com

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Scientific Tracks & Abstracts

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Sessions

Neurology | Neuroscience | Neurological Disorders | Neurotherapeutics

Session Chair
Zaiga Kalnberza-Ribule
National Rehabilitation Center Vaivari | Latvia

Session Introduction

Title: **Novelo-Merida: The first Mexican Physiotherapy method based in Neuroscience**
Novelo Pérez Fernando | Neurodevelopment center | Mexico

Title: **The diagnostic yield of Hypotonia investigations: A 10-year single center study**
Mohammed Hassan AlBanji | King Abdulaziz University | Saudi Arabia

Title: **The mediator effect of cognition on the relationship between Brain lesion location and Dysphagia in patients with Stroke: Applying a structural equation model**
Shiva Ebrahimian Dehaghani | Shiraz University of Medical Sciences | Iran

Title: **A case report of Myasthenia Gravis treated with Plasmapharesis**
Theresia Christin | Sriwijaya University | Indonesia

Novelo-Merida: The first Mexican Physiotherapy method based in Neuroscience

Fernando Novelo Pérez
Neuro Development Center(NDC), Mexico

Pediatric Physiotherapy (PF) is in constant evolution, in the last 20 years neuroscience has made new discoveries and advances about the nervous system and how babies and children learn to move, how the nervous system works after a lesion and what neuroplastic strategies should activate during therapy. These new knowledge is impacting directly in every theory and techniques in the PF world. Mexico does not have proper strategies to satisfy the need of babies and children with neurological damage, congenital and/or genetic anomalies, Mexican still use classic physiotherapy without scientific evidence; the physiocognitive method Novelo-Merida (NM) is the Mexican proposal of how to interpretate and apply all these knowledge under the optic of the cognitive theory. Fig 1. The NM method solves basic questions that every physiotherapist who works with these children should propose during the planning and intervention of the therapy, it demonstrates with scientific evidence how this method plays a determinant role during the cognitive development in the first years of life, it also highlight the importance of activate neuroplastic process as the neuronal organizers of the higher mental functions to achieve an effective and evolved motor skills. This method is designed with a very accessible methodology perfect for teaching in universities at a low cost and most important made by Mexican instructors.

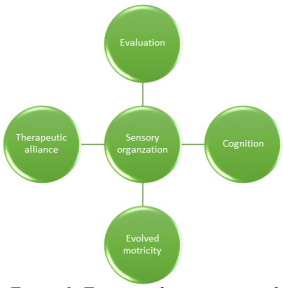


Figure 1. Functional organizational diagram of the NM method. 2018

Biography

Fernando Novelo Pérez, Director and senior therapist of NDC a cognitive pediatric physical center and author of the fisiocgnitive Novelo-Merida method. Pediatric physical therapist with 38 years of experience, bachelor´s degree in psychology, master´s degree in pedagogy, Pioneer in the pediatric physiotherapy in the south of Mexico. College professor at the university Marista of Merida, coordinator and professor of post graduate courses in pediatric physiotherapy at the Marista University.

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The diagnostic yield of Hypotonia investigations: A 10-year single center study

Mohammed Hasan Albanji[1], Aqeela Al-Hashim[2], Riyam AlAnazi, Zahra Aleisa, Abdulaziz AlSaad and **Dalya Alam**
[1]King Abdulaziz University, Saudi Arabia
[2]King Fahad Medical City, Saudi Arabia

Introduction: The hypotonic neonate represents a diagnostic challenge as a lesion at any level in the neuro-axis may cause hypotonia. Distinguishing among these pathologies is a particularly difficult task. Identification of the cause is essential for determining prognosis, associated morbidities, and recurrence risk.

Aim: We aim to study the diagnostic yield of the investigations commonly used in hypotonia work up. Methods and Patients: a cohort retrospective study over a period of 10 years at King Fahad medical city, a tertiary care center in the central region of Saudi Arabia. Patients charts were collected from electronic patient records.

Results: 1592 patients' charts were retrieved and screened. 164 patients fit the inclusion criteria of the study and among those 129 patients had MRI that were reviewed. Center hypertonia accounted for 50%(83), peripheral hypotonia 38%(17) and mixed hypotonia 31%(52) of patients. 54.9%(90) of Patients age ranged between 12-60 months at presentation. 88% (n 146) of patients associated with abnormal history and examination. Molecular testing was performed for 44% (74) of patients. Total of 65 Microarray studies were done, 27%(18) of these studies reported as abnormal and 9%(6) microarrays were diagnostic. 55 gene panels were done, 72%(44) of them were abnormal and 30%(17) were diagnostic. Total of 53 targeted genes were done, 56%(30) were abnormal and 39%(21) were diagnostic. Total of 61 whole exome sequencing were done, 75%(37) were abnormal and 68%(42) were diagnostic. A total of 126 MRI were reviewed by a neuro-radiologist, 56%(88) of MRI reported as abnormal and 33%(42) contributed to the diagnosis.

Conclusion: Various molecular genetic testing especially whole exome sequencing is our recommended next step in the diagnosis of patients with hypotonia. Whole exome sequencing has evolved to be more cost effective and time saver for the diagnosis of hypotonic patients. Karyotyping and Metabolic work up yield is disappointingly low.

Biography

Mohammed Hasan Albanji, graduated from King Abdulaziz University in 2013 with bachelor of medicine and surgery (MBBS), then obtained his Saudi and Jordanian Board of pediatric Neurology last 2018. He has great interest and passion in medical research in the field of complex pediatric neurological diseases and pediatric movement disorders. Himself and his group are focusing on studying the population of western part of Saudi Arabia with the aim to have a better understanding of the burden of child neurological disease in our region and formalize a regional health care plans and guidelines according to the available resources.

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The mediator effect of cognition on the relationship between Brain lesion location and Dysphagia in patients with Stroke: Applying a structural equation model

Shiva Ebrahimian Dehaghani
Shiraz University of Medical Sciences, Iran

Background: Cognitive status is important for performing the assessment and treatment of dysphagia. Nonetheless, the cognitive neuropsychological profile of patients with stroke and dysphagia is not studied as deeply enough as it should be. On the one hand, focal and non-focal brain lesions may lead to dysphagia and cognitive disorders, and on the other hand, the cognitive status possibly affects swallowing. In this study, cognition is supposed to be a mediator between brain lesion and swallowing disorder (dysphagia). The role of cognition and attention as mediators between brain lesion and dysphagia was evaluated via three causal models in which the causal mechanisms of swallowing were explained.

Methods: Eighty-eight patients with their first stroke (34 women and 54 men) in the acute phase (mean: 3.5 days after stroke; SD: 2.7) participated in this research. The data of dysphagic and non-dysphagic patients were entered into structural equation models. Two relationships were estimated: a direct relationship between brain lesion and dysphagia and an indirect association between brain lesion and dysphagia through cognition and attention.

Results: The goodness-of- fit indices confirmed the three models. Our first model proved a good fit [CFI = 1.00; TLI = 1.00]. The second model revealed an appropriate goodness of fit [CFI = 1.00; TLI = 1.00]. Our third model also showed a good fit [CFI = 1.00; TLI = 1.00].

Conclusion: It is suggested that in the assessment and treatment of dysphagia, cognition is better to be considered as a mediator along with physical aspects of dysphagia.

Biography

Shiva Ebrahimian Dehaghani, assistant professor, active clinician and researcher. She has been a medical speech-language pathologist for over 20 years and has worked evaluating and treating communication and swallowing disorders across multiple patient populations from NICU to stroke and geriatrics. She earned her bachelor's degree in Speech-Language Pathology from the Isfahan University of Medical Sciences (IUMS) in Isfahan, Iran in 1999. She went on to earn her Master's degree in Speech-Language Pathology from the University of Social Welfare & Rehabilitation Sciences (USWR) in Tehran, Iran in 2002. After finishing her Master's degree, she became a researcher, active clinician, lecturer, and clinical supervisor in various clinical and educational settings in Iran. In 2009, she was employed by Shiraz University of Medical Sciences as instructor. Then, in 2011, she began his Ph.D. in Communication Sciences and Disorders at USWR in Tehran, Iran. She completed her doctoral work with a primary research and teaching focus revolving around neurogenic dysphagia in 2016.

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A case report of Myasthenia Gravis treated with Plasmapharesis

Theresia Christin
Sriwijaya University, Indonesia

Background: Myasthenia gravis is a neuromuscular autoimmune disorder. The most common form is muscle weakness that is not accompanied by pain, weakness is exacerbated by activity and improves when resting. Exacerbations and remissions can occur, especially in the first years of the disease. Remission rarely occurs complete or permanent.

Case Report: A 35-year-old woman was treated in the Neurology Department of RSMH due to being unable to eat and drink due to not being able to swallow slowly. The diagnosis of myasthenia is made primarily from clinical presentation, which is based on history taking and neurological physical examination and continued with additional work up. In electrophysiological examination, characteristic of myasthenia which is the rapid reduction of amplitude in CMAP during serial repetitive stimulation on peripheral nerve at frequency of 3/ second was found. Previous diagnosis have been made through ENMG with Harvey Masland positive without thymoma (normal chest CT scan). Although a definitive diagnosis is obtained by examination of antiacetylcholine receptor antibodies, using repetitive nerve stimulation is quite specific and indicates a high likelihood that this patient is diagnosed with myasthenia gravis. Clinically, patients are categorized through the clinical classification of MGFA as Class IIIb, where the patient mainly affects the oropharyngeal muscles with a moderate weakness. During treatment the patient was given corticosteroids and antiacetylcholinesterase. The choice of corticosteroids and antiacetylcholinesterase as therapy clinically provides improvement in patients.

Conclusion: Myasthenia gravis is a fluctuating disease. Clinical repair and worsening can occur. Exacerbation of myasthenia is a general term that can be defined as an increase in the degree of weakness in the bulbar muscles, breathing, or muscles of the arms and legs. Both conditions of this crisis show almost the same clinical appearance. The choice of therapy and dosage must be adjusted to each individual's condition.

Biography

Theresia Christin, Neurologist at Siloam Sriwijaya Palembang. He completed his Neurology Specialist education at the Faculty of Medicine at Sriwijaya University, Palembang. In addition to being a doctor, he also teaches general medicine studies at Sriwijaya University. He is a member of the Indonesian Neurologist Specialist Association. His health services include: Dementia Management, Aneursym Coiling Migraine Treatment, Spina Bifida Consultation, Laser Therapy for Bell's Palsy, Stroke Consultation, Neuromodulator, Parkinson's Consultation, Epilepsy Treatment, Brain Angiography (DSA), Consultation for Nerve Disease, Parkinson's Treatment, Spinal Injection Therapy.

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Poster

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Clinical and diagnostic values of the determination of CSF specific proteins in Alzheimer's Disease patients

Alessandro Rabbito[1], Agnieska Kulczynska-Przybik[1], Pawel Muszynski[1], Bartolomiej Borawski[2], Agnieska Slowik[2], Barbara Mroczko[1]
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Statement of the Problem: Alzheimer's disease (AD) is the most common dementia that nowadays affected more than 30 million of people in the world and is estimated that in 2050 the number of people affected by AD will triple. AD is one of the biggest challenges for modern neuroscience and medical diagnostics, because to diagnose AD is needed to perform neurobiochemical, neuroimaging and neuropsychological analyses. For this reason AD diagnosis is very difficult in the early stage of the disease.

Methodology & Theoretical Orientation: The objective of this project is to detect and asses the pathological concentration of specific protein in peripheral fluid samples to obtain novel biomarkers to diagnose Alzheimer's disease. ELISA assay was used to determine the concentrations of specific proteins in CSF of patients with Alzheimer's disease, and in control groups.

Findings: Chemokines can be involved in AD with their pro-inflammatory or anti-inflammatory ability. In the first step we focused our work on CXCL8 and its receptor CXCR2 and we measured the concentrations of these proteins in AD patients, mild cognitive impairment patients (MCI) and in a control group. In these preliminary results was finding a statistical difference in CXCL8 concentration between AD patients and control group but not between AD patients and MCI patients. No statistical differences were found in CXCR2 concentration.

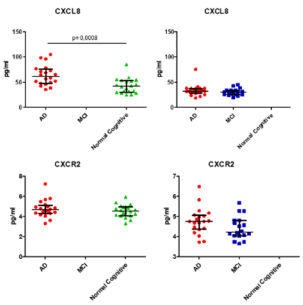


Fig. 1. CXCL8 and CXCR2 CSF concentration. Comparison between Alzheimer's disease patients (AD), mild cognitive impairment patients (MCI) and normal cognitive people as control group.

Conclusion & Significance: These are the preliminary results of what should be a study that aims to evaluate the potential of different proteins as biomarkers for AD. These first data shown that CXCR2 concentration in CSF does not change in the different condition considered. Further CXCL8 experiments will be performed to confirm the data.

Biography

Alessandro Rabbito, from Naples, Italy, PhD student at Medical University of Bialystok in the Neurodegeneration Diagnostic Department, with the supervision of Professor Barbara Mroczko. Studied Medical Biotechnology at Università degli Studi di Napoli Federico II, graduated in 2017 summa cum laude. As an undergraduate student Worked in the Endocannabinoid Research Group in Italy for two years. Interested in endocannabinoids and endocannabinoid system. Goal is to investigate and unrevealing the endocannabinoid system role in neurodegenerative diseases.

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Hearing screening of infants: BERA values, guidelines, and comparative programs

Waleed B Shuaib
Kuwait University, Saudi Arabia

Statement of the problem: In Kuwait, routine audiological assessments of referred children revealed that, out of the children suspected for hearing loss, 42% actually had some level (mild-moderate) of hearing loss. Establishment of a hearing screening test of newborns in Kuwait was warranted to test hearing loss; it was necessary to examine comparative programs in developed countries.

Purpose of study: The aim of the study was two-fold, first, to detect permanent hearing loss in the UNHS program at KOC hospital in Kuwait. Second, to provide guidelines to the newly established UNHS program in Kuwait, considering the current issues & guiding principles in an international setting.

Findings: First aim, the incidence of permanent hearing loss was different across nursery levels. In the "well baby" group, 1 % had profound sensorineural hearing loss, 1 % had severe (70 dBnHL) sensorineural hearing loss, and 98% had normal hearing level. In the "high-risk" group, 26.67 % had profound sensorineural hearing loss, 20 % had moderate (60 dBnHL) sensorineural hearing loss, and 53.33 % had normal hearing level.

Second aim, we provide detailed guidelines for UNHS in Kuwait: screening, diagnosis, and intervention; at birth: newborn screening, 1 month of age: screening completed, 3 months of age: diagnostic evaluation, 6 months of age: intervention services provided.

Conclusions & significance: We have established detailed and specific guidelines for a successful UNHS program, to ensure: -babies who have a diagnosed permanent hearing loss enroll in early intervention including hearing aid fitting, speech and language therapy, by 6 months of age. -All infants who do not pass the screening will have a diagnostic audiologic evaluation before 3 months of age. -All infants identified with hearing loss receive appropriate early intervention services before 6 months of age. A data management system is maintained to document hearing screening and diagnostic audiological evaluation results and intervention services.

Biography

Waleed B Shuaib is a neuroscientist with special interest in clinical neurophysiology.

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Endocannabinoidome Tone in Multiple Sclerosis patients

Alessandro Rabbito, Michał Biernacki, Alina Kułakowska, Joanna Tarasiuk, Elzbieta Skrzydlewska and **Barbara Mroczko**
Medical University of Bialystok, Poland

Statement of the Problem: Multiple sclerosis (MS) is an inflammatory demyelinating disease of the central nervous system, caused by an autoimmune response against myelin that leads to progressive neurodegeneration and disability. Although recent studies have provided new data on molecular mechanism underlying the onset of the disease, there is still a gap in the knowledge about lipid derivatives participating in development of MS. In this context, endocannabinoid system may be important. Endocannabinoids are a class of bioactive lipids derivatives, among which AEA and 2-AG, are the most abundant endocannabinoids in human body and the first ones to have been discovered. Beyond these two major endocannabinoids there are other related compounds considered as endocannabinoids and endocannabinoids-like molecules, members of a more big family of lipid derivatives molecules that compose the endocannabinoidome. Considering that, one of the most interesting properties probably is the entourage effect, several studies reported that these molecules can enhance endocannabinoids levels/biological activity. Furthermore, these bioactive lipids are not just entourage molecules, but reveal also functions by activating different receptors.

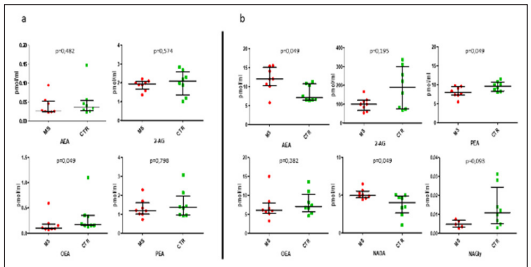


Figure 1. Major endocannabinoids and endocannabinoids-like molecules concentration in CSF (a) and serum (b)

Methodology & Theoretical Orientation: The aim of this studies was investigate the dysregulation of endocannabinoidome tone, in biological fluids as CSF and serum, in MS patients. The concentrations of endocannabinoids and endocannabinoid-like molecules were assessed, after SPE lipid extraction, using ultra-performing liquid chromatography tandem mass spectrometry in positive-ion mode.

Findings: These preliminary results obtained analyzing a panel of more than 10 endocannabinoids and endocannabinoid like molecules, confirm that there is an alteration in endocannabinoidome tone in MS.

Conclusion & Significance: Although several studies reported changes in endocannabinoids tone in neurological/neurodegenerative diseases, but there are very few papers concerning a big panel of endocannabinoid like molecules in serum and CSF of MS patients. Because the alterations are different between molecules, further investigation with a bigger group of patients are needed.

Biography

Alessandro Rabbito, from Naples, Italy, PhD student at Medical University of Bialystok in the Neurodegeneration Diagnostic Department, with the supervision of Professor Barbara Mroczko. Studied Medical Biotechnology at Università degli Studi di Napoli Federico II, graduated in 2017 summa cum laude. As an undergraduate student Worked in the Endocannabinoid Research Group in Italy for two years. Interested in endocannabinoids and endocannabinoid system. Goal is to investigate and unrevealing the endocannabinoid system role in neurodegenerative diseases.

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Brainstem Auditory Evoked Potentials (BAEP) during development and aging

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Statement of the problem: The brainstem auditory evoked potentials (BAEP) is an effective method of evaluating the auditory pathway from the cochlea to the brainstem. BAEP is a useful technique in the diagnosis of neurological and audiological disorders. However, appropriate BAEP interpeak intervals should be established for age-group,s for clinical testing of patients.

Purpose of study: The purpose of this study was to determine BAEP peak latencies (I, III, V) and interpeak intervals (I-III, III-V, I-V) in newborns (10-80 days), children (1-10 years), adults (18-40 years) & (41-60 years), old subjects (63-80 years) so that they can be used in Ahmadi hospital and in Kuwait.

Methodology: A Nicholet Viking IV system was used to record BAEPs in a quiet room. The headphone delivered click stimulation (square pulse of 0.1 msec duration) to the tested ear (70 dB above click perception threshold). 1000 stimulations were applied, with a repetition rate of 11.1/sec.

Findings: The main results of our study concerning peak latencies (I, III, V) and interpeak intervals I-III, III-V, I-V were as follows:- Newborn peak latencies and intervals were the longest compared to all other groups. - Age-groups 1-10 years, 18-40 years, and 41-60 years had similar peak latencies and similar interpeak intervals.- Old group 63-80 years peak latencies and intervals were slightly longer than these three age-groups. -Male and female BAEP values were similar (no significant difference). This was the case for adult (18-60 years) and older subjects (63-80 years).

Conclusions & significance: We have established appropriate upper limits of interpeak intervals for newborns (10-80 days), children (1-10 years), adults (18-60 years), and older subjects (63-80 years). This will enable to distinguish between normal and abnormal interpeak intervals in a given subject according to their age group.

Biography

Eesa W Alshuaib is a Graduate student at the National University Ireland Galway, in Medical Science with special interest in Neuroscience.

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A case report of Myasthenia Gravis treated with Plasmapharesis

Theresia Christin
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Background: Myasthenia Gravis is a neuromuscular autoimmune disorder. The most common form is muscle weakness that is not accompanied by pain, weakness is exacerbated by activity and improves when resting. Exacerbations and remissions can occur, especially in the first years of the disease. Remission rarely occurs complete or permanent.

Case Report: A 35-year-old woman was treated in the Neurology Department of RSMH due to being unable to eat and drink due to not being able to swallow slowly. The diagnosis of myasthenia is made primarily from clinical presentation, which is based on history taking and neurological physical examination and continued with additional work up. In electrophysiological examination, characteristic of myasthenia which is the rapid reduction of amplitude in CMAP during serial repetitive stimulation on peripheral nerve at frequency of 3/ second was found. Previous diagnosis have been made through ENMG with Harvey Masland positive without thymoma (normal chest CT scan). Although a definitive diagnosis is obtained by examination of antiacetylcholine receptor antibodies, using repetitive nerve stimulation is quite specific and indicates a high likelihood that this patient is diagnosed with myasthenia gravis. Clinically, patients are categorized through the clinical classification of MGFA as Class IIIb, where the patient mainly affects the oropharyngeal muscles with a moderate weakness. During treatment the patient was given corticosteroids and antiacetylcholinesterase. The choice of corticosteroids and antiacetylcholinesterase as therapy clinically provides improvement in patients.

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Theresia Christin, Neurologist at Siloam Sriwijaya Palembang. He completed his Neurology Specialist education at the Faculty of Medicine at Sriwijaya University, Palembang. In addition to being a doctor, he also teaches general medicine studies at Sriwijaya University. He is a member of the Indonesian Neurologist Specialist Association. His health services include: Dementia Management, Aneursym Coiling Migraine Treatment, Spina Bifida Consultation, Laser Therapy for Bell's Palsy, Stroke Consultation, Neuromodulator, Parkinson's Consultation, Epilepsy Treatment, Brain Angiography (DSA), Consultation for Nerve Disease, Parkinson's Treatment, Spinal Injection Therapy.

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e-Poster

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Reduction of seizure induction and death in susceptible Autistic Disorders

Maria V Tejada-Simon
University of Houston, USA

Fragile X Syndrome (FSX) is the leading single gene cause of autism and intellectual disability (ID). Recent studies report a high expression of Rac1 in mice and humans with FXS, characterized, among other phenotypes, by cognitive impairment and incidence of audiogenic seizures upon presentation with loud noises. Our laboratory has previously shown that pharmacological inhibition of Rac1 with a particular inhibitor, NSC23766, decreases cognitive impairment in FXS. In an effort to further evaluate whether this treatment rescues the FXS-associated seizure phenotype, an independent cohort of animals, naïve to any type of behavioral manipulation, was pharmacologically treated with NSC23766. This treatment resulted in the reduction of both audiogenic seizures and death in FXS mice. These findings suggest that small GTPase hyperactivity may play an important role in FXS molecular pathology, contributing to the phenotypic alterations usually described in the literature.

Biography

Maria V Tejada-Simon is an Associate Professor of Pharmacology, Biology and Psychology, and member of the Biology of Behavior Institute at the University of Houston in Houston, Texas. Current research in her laboratory examines the role of small GTP-binding proteins in cognition, with emphasis in mental retardation, schizophrenia, neurodegenerative as well as autistic disorders.

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The state of stress in today's leadership world

Asha Mankowska
Your Favorite Business Coach, USA

Motivation: Stress has become a major predicament in business and leaders are increasingly susceptible to it. Stress has been implicated as an important determinant of leadership functioning. Their stress is proven to impact their followers (companies, countries, team members) and if not prevented or managed properly, it can result in anxiety, depression, and as a direct consequence: burnout, or worse. CEOs and management face many challenges, and critical failures and overwhelming odds can easily break anyone down and make them lose sight of their goals. They have to have the skills to overcome stressful situations and demonstrate conduct that will make a business, an organization or country, productive and profitable.

The Problem: A leader's stress level will influence his or her behavior and that can impact the stress levels and potential for burnout in subordinates. A lack of resources and time are the most stressful demands experienced by leaders. Stress is caused by trying to do more with less, and to do it faster. For 88% of leaders, work is a primary source of stress in their lives, and having a leadership role increases the level of stress. Unfortunately, very few (only 28%) companies provide tools to help management deal with stress more effectively.

Methodology Study & Results: During the many years, I have worked with business leaders, I have come to recognize the 7 most common stressor determinants and I have developed methodology to conquer them. The top 7 sources are: 1. Expected High Performance in a Short Period of Time, 2. Strong Brand Loyalty, 3. Conflict Management, 4. Reputation Management, 5. Hiring the Right Team, 6. Professional Obligations vs. Balanced Life, 7. Lack of Support And Accountability; The new avenues for stress prevention and management (individual versus corporate approach) are using stress as an efficient fuel: 1. Using certain technology, online media platforms, aps, to increase productivity, efficiency, and profits as well as leaders' wellbeing and confidence. 2. Creating strong bonds with clients using innovation and differentiation, that results in full control over the process. 3. Address & control conflict with authority, even before it arises. 4. Learning strong communication with the right infrastructure and teamwork. 5. Mastering the art of delegation & hiring the right people to maximize efficiency. 6. Demonstrating healthy & balanced conduct as an example for the company. (Vacation, hiking conferences, team building, wellbeing) 7. Establishing support & accountability systems in place outside of corporate environment (masterminds, mentors, coaches, trainings, business conferences) These case studies were based on my clients' feedback and their results achieved within less than one year of their solid commitment, execution and implementation of the proposed methods used in my business practice.

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Enteric short chain fatty acids: Microbial modulators of Mitochondria, metabolism and mind: Implications in Autism Spectrum Disorders

Derrick F MacFabe
Kilee Patchell-Evans Autism Research Group (KPEARG), Canada

Clinical observations suggest that gut and dietary factors, transiently worsen, and in some cases appear to improve, symptoms in autism spectrum disorders (ASDs), but the reason of this is unclear. Recent evidence suggests ASD as a family of systemic disorders of increasing incidence manifesting altered immunity, metabolism and gene expression, and altered components of the gut microbiome. Pre or perinatal infection, antibiotics, hospitalization, diet or recent human migration are emerging as a major risk factor for ASD and many other neurological conditions. Can a common environmental agent link these disparate findings? Dr. MacFabe has proposed that short chain fatty acids (SCFA) represent a group of compounds produced by the host microbiome from dietary carbohydrate fermentation, which can induce widespread effects on host gut, brain, immunity and behavior that are time and dose sensitive. Collectively SCFA provide a mechanism where the microbiome may "sculpt" brain development and behavior. Imbalances in SCFA metabolism may link the diverse symptoms and findings in ASDs and related conditions.

Collectively, SCFA metabolism alterations are consistent with the symptoms and proposed underlying mechanisms of ASDs, and offer many novel dietary nutritional, and metabolic interventions that may be useful in the possible prevention and treatment in these and other neurological conditions (anxiety/depressive disorder, ADHD,OCD, eating disorders, IBS, Parkinson, stroke, neurotrauma, dementia). Collectively, this research offers further support that gut microbiome metabolites, such as dietary or enteric bacterially produced SCFA are: 1) underappreciated modulators of brain development, function and behavior that are time, tissue and dose specific, 2) may be plausible environmental agents that can trigger and possibly prevent ASDs, 3) offer potential novel preventative and therapeutic therapies throughout the lifecycle, and 4)deserve further exploration in basic science, agriculture, nutrition and clinical medicine.

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The use of Ultra-Flexible Dynamic Bracing to improve active stabilty, balance, movement and tone control for improved active function

Nancy Hylton
Children's Therapy Center, USA

The purpose of this type of bracing has been to form an ultra-flexible exoskeleton for improved active stability, balance, and guiding of movement in order to reduce compensatory hypertonus through improved sensory awareness, easier access to CPG functions and adaptive movement control and balance. We discovered that certain materials with greater rebound memory provide more assistance to sensory body awareness and motor learning through return to a more midline biomechanical orientation after movement away from midline in a joint or body part. A very old plastic, polypropylene when stretched very thin proved to have ideal memory for foot/ankle bracing function. Our long-standing partnership with Don Buethorn, CPO and Cascade P and O (now Cascade DAFO) allowed creative development of many unique orthotic products. Very thin polyethylene was used in hand/ wrist, knee extension and trunk bracing to permit some some small triplanar movements to occur for invitation to active movement. Medium density foam with a quick rebound proved best for foam custom foam construction using an electric carving knife. The development of numerous 4 mm thick Neoprene support systems, initially thought to be primarily providing primarily joint and soft tissue stability through compression was furthered with a partnership with Dave Baumgardner and the Benik Corporation. Evolution into Lycra came through a very creative partnership with a CTC parent, Cheryl Allen, trying to help her adopted children. SPIO Compression is now helping children and adults to move more easily in the US and in many countries around the world.

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Alcohol in the aging brain – The interplay between Alcohol Consumption Cognitive Decline and the Cardiovascular System

Melinda A Mende
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As our society grows older new challenges for medicine and healthcare emerge. Age-related changes of the body have been observed in essential body functions, particularly in the loco-motor system, in the cardiovascular system and in cognitive functions concerning both brain plasticity and changes in behavior. Nutrition and lifestyle, such as nicotine intake and chronic alcohol consumption, also contribute to biological changes in the brain. This talk addresses the effect of alcohol consumption on cognitive decline, changes in brain plasticity in the aging brain and on cardiovascular health in aging.

Thus, studies on the interplay of chronic alcohol intake and either cognitive decline or cognitive preservation are outlined. Because of the inconsistency in the literature of whether alcohol consumption preserves cognitive functions in the aging brain or whether it accelerates cognitive decline, it is crucial to consider individual contributing factors such as culture, health and lifestyle in future studies.

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Therapeutic ketosis and the broad field of applications for the ketogenic diet: Ketone ester applications & clinical updates

Raffael Pilla
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It has been recently shown that nutritional ketosis is effective against seizure disorders and various acute/chronic neurological disorders. Physiologically, glucose is the primary metabolic fuel for cells. However, many neurodegenerative disorders have been associated with impaired glucose transport/metabolism and with mitochondrial dysfunction, such as Alzheimer's/Parkinson's disease, general seizure disorders, and traumatic brain injury. Ketone bodies and tricarboxylic acid cycle intermediates represent alternative fuels for the brain and can bypass the ratelimiting steps associated with impaired neuronal glucose metabolism. Therefore, therapeutic ketosis can be considered as a metabolic therapy by providing alternative energy substrates. It has been estimated that the brain derives over 60% of its total energy from ketones when glucose availability is limited. In fact, after prolonged periods of fasting or ketogenic diet (KD), the body utilizes energy obtained from free fatty acids (FFAs) released from adipose tissue. Because the brain is unable to derive significant energy from FFAs, hepatic ketogenesis converts FFAs into ketone bodies-hydroxybutyrate (BHB) and acetoacetate (AcAc)-while a percentage of AcAc spontaneously decarboxylates to acetone. Large quantities of ketone bodies accumulate in the blood through this mechanism. This represents a state of normal physiological ketosis and can be therapeutic. Ketone bodies are transported across the blood-brain barrier by monocarboxylic acid transporters to fuel brain function. Starvation or nutritional ketosis is an essential survival mechanism that ensures metabolic flexibility during prolonged fasting or lack of carbohydrate ingestion. Therapeutic ketosis leads to metabolic adaptations that may improve brain metabolism, restore mitochondrial ATP production, decrease reactive oxygen species production, reduce inflammation, and increase neurotrophic factors' function. It has been shown that KD mimics the effects of fasting and the lack of glucose/insulin signaling, promoting a metabolic shift towards fatty acid utilization. In this work, the author reports a number of successful case reports treated through metabolic ketosis.

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The Role of malfunctional Mitochondria in the development of Parkinson's Disease

Hari Pai
King's College London, UK

Statement of the Problem: Parkinson's' Disease is the second most common neurodegenerative disorder in the world. It is thought to occur due to degradation of dopaminergic neurons within the substantia nigra pars compacta of the basal ganglia. This paper elucidates on a theory that one potential reason for Parkinson's developing is due to problems with mitochondria.

Methodology & Theoretical Orientation: A literature review of 2 web databases (PubMed, Web of Science) yielded the papers which were used in this review.

Findings: The problems with mitochondria are thought to come by a variety of factors. Factors offered in this paper include the mutation or absence of parkin or PINK1 genes. This alteration in parkin and PINK1 leads to changes in the mitochondria which are present in the human body. These can lead to compromised complex activity and increased oxidative stress. Increased oxidative stress (via free radicals or reactive oxygen species) can lead to deletion of mtDNA (with mitochondria having its own genome). The deletion of mtDNA is a problem as it is known to be one of the factors leading to Parkinson's.

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Metamemory in primary Insomnia

Paula Pedic
University of Zadar, Croatia

Aristotle once said that memory is the scribe of the soul. Memory indeed is one of the core cognitive abilities in human beings and as such, it is important to preserve it. It has been shown that stabilization of memory traces is closely related to sleep. If sleep really does play a role in memory consolidation, the question that arises is: what happens to memory if sleep is distorted? One of the most common sleep disorders is primary insomnia. It has been shown that memory of patients with primary insomnia is impaired, especially after interfering tasks. However, metamemory beliefs of those patients still remain unexamined. That is why the focus of this study was to reveal the metamemory beliefs of those students who suffer from primary insomnia, compared to healthy students. After an entrance examination, all of the subjects filled in Athens scale questionnaire and were then asked to learn a declarative memory task. Subject's overnight memory change was tested in the morning, followed by metamemory beliefs examination. It was hypothesized that, compared to healthy subjects, students who suffer from primary insomnia will overestimate their declarative memory abilities. Metamemory calibration (correlations between memory predictions and performance) was examined in both, healthy subjects and subject with primary insomnia. The results indicated that calibration efficiency really was decreased in subject with primary insomnia.

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The role of Bilateral Repetitive Trans Cranial Magnetic Stimulation in Stroke Patients

Ann Ali Abdelkader Hanafy
Cairo University Hospital Medical School, Egypt

Repetitive transcranial magnetic stimulation is a noninvasive brain stimulation to the motor cortex. It can alter excitability of the brain after stroke by modulating cortical activity. At low frequencies (1 HZ or less), it has an inhibitory effect, whereas at high frequencies (5-25 HZ) (Chen et al., 2008). Fifty-five patients with subacute stroke (2 weeks to 6 months) were divided into four groups: bilateral, inhibitory, stimulatory and control groups. Bilateral group received five sessions of high-frequency 5Hz rTMS over the affected hemisphere alternating with low-frequency 1 Hz rTMS over the unaffected hemisphere. Inhibitory group received low frequency 1 Hz rTMS over the unaffected hemisphere alternated by sham 5 Hz rTMS over the affected hemisphere. Stimulatory group received high frequency 5 Hz rTMS over the affected hemisphere alternated by sham 1 Hz rTMS over the unaffected hemisphere. Control group received sham stimulatory rTMS over the affected hemisphere alternated by sham inhibitory rTMS over the unaffected. All groups showed statistically significant improvement of the motor disability of the paretic upper limb post stroke assessed by FMA, WMFT and change in excitability assessed by MEP, from before to after sessions and after one month. When groups were compared with each other, bilateral group showed the best improvement of spasticity measured by the modified ashworth scale, while all other groups failed to change the Ashworth scale. Control group failed to show change in the MEP and stimulatory group failed to change the MEP of the healthy hemisphere and its effect was on the unhealthy hemisphere only.

Conclusion: According to the finding yielded from this study, it can be concluded that five daily sessions of bilateral rTMS combined with one month of task-oriented physiotherapy, improves motor disability of the paretic upper limb after stroke. Inhibitory and stimulatory rTMS show nearly same efficacy as bilateral protocol, however bilateral stimulation is superior in spasticity.

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Effects of Policosanol on Post-Stroke Cognitive Impairment: A case report series study

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Ischemic stroke is a leading cause of disability, including post-stroke cognitive impairment (CI), for which no effective therapy is available. Post-stroke patients are managed with antiplatelet drugs, and antihypertensive, hypoglycemic and/or cholesterol-lowering drugs, as needed. Policosanol is an antiplatelet and cholesterol-lowering agent. Previous studies demonstrated that adding policosanol to aspirin (AS) therapy improves post-stroke neurological recovery compared to placebo + AS. Policosanol effects on post-stroke CI have not been investigated yet. This study was undertaken to record the evolution of recent post-stroke (≤30 days from onset) survivors with CI treated with policosanol + AS for 12 months. Patients with recent post- stroke CI untreated with policosanol were enrolled, managed according to guidelines and started on policosanol (20 mg/day) and AS (125 mg/day) for 12 months. Routine neurological examinations; control of therapy compliance and adverse experiences (AE) were done. CI was assessed with the Luria-Nebraska test at baseline and at 12 months. Stable condition was pre-defined as no change on test results, improvement as changes to better levels and deterioration as changes to worst levels. Patients (56) (37 men, 19 women) (73 years) exhibited vascular risk factors: hypertension (76.8%); dyslipidemia (50.0%); smoking (26.8%); and diabetes (17.9%). All patients completed the follow-up and none had a recurrent vascular event. Only 1 patient (1.8%) had further CI deterioration; 27 (48.2%) remained stable, and 29 (51.8%) exhibited mild or moderate improvement. Treatment was well tolerated. Only three patients reported mild AE. It is concluded that patients with CI post-ischemic stroke treated with policosanol + AS for 12 months had good evolution since none died, none had recurrent events, and most experienced CI improvement or remained stable. This study has limitations for stronger conclusions since it is a case report series, but our results encourage to investigate such effects in randomized, double-blind, placebo-controlled studies.

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Adopting laughter therapy to get dosage of happy hormones to remove stress caused by being in slight pain, being depressed, being unhappy anxious or sad. saying positive affirmations aloud changes body cell energy

Suchi Spore
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Statement of the Problem: There is a lack of awareness about what happy hormones are, how to use positive words to feel energetic and what can be done to get happy hormones. People tend to feel unhappy for multiple reasons and neuropathic pain adds on Stress levels of not only the patient but the caregivers as well. Being in pain leads to feeling depressed and anxious in some cases.

Methodology & Theoretical Orientation: Review of Books and Research shows that getting a dosage of happy hormones will not only ease slight pain of the patient but feeling happy will also have a positive impact on the recovery of the patient. Adopting Laughter therapy and getting hormones which makes one feel good will help many to recover from Neuropathic pain /Long term sadness caused by having grief, Anger or Resentment, Depression & Anxiety.

Findings: One needs to work on his/her energies using Laughter Therapy which is a positive approach for not having Depression & Anxiety caused by Neuropathic pain. The therapy can be used as a Holistic way to recovery.

Conclusion & Significance: The Laughter therapy which includes ways to get the dosage of happy hormones promotes overcoming Depression & Anxiety caused by Neuropathic pain, is a fun way to manage pain. Repeated sessions to be conducted to remind patients that life while having pain or during the recovery should go beyond just seeking medical and counselling help and also include rebuilding Spiritual, Physical, Emotional, Relational and Mental health. The model has been put together from for testing in many settings including hospitals, elderly homes and senior citizen centres. This is not a research book or paper. It is just an effort to demystify the help available for Depression & Anxiety caused by pain. It is an attempt to motivate and encourage people to seek help and take a simple approach to remember and work on all aspects of their recovery.

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Brain Insulin Resistance in Alzheimer's Disease: Targeting phosphoinositide 3-kinase (PI3K)/Akt/GSK-3β pathway

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ICV-STZ was used for the model of sporadic Alzheimer's disease being established. Adult male Wistar rats (48) weighing 200-300 g bred in Central Animal House facility of Panjab University were used. Animals were randomly divided into 8 groups comprising 6 animals in each group as follows: Protocol lasted for 21 days, sacrificing animals on 22nd day followed by isolation of serum and dissection of cortex and hippocampus, preserving the same for further analysis. Behavioral studies like Morris water maze was done for assessing spatial memory, novel object recognition for associative memory and actophotometer was performed for locomotor activity. Biochemical estimations for antioxidant activity or oxidative stress such as reduced glutathione estimation, superoxide dismutase assay, catalase assay, glutathione peroxidase assay, myeloperoxidase assay, glutathione S-transferase assay, lipid peroxidation assay, and protein carbonylation assay were performed in the homogenates of cortex and hippocampus of the brain which are the specific regions for memory, learning and cognition. For nitrosative stress, nitrite estimation was done. Protein concentrations were determined by the biuret method. Cholinergic activity was evaluated by acetylcholinesterase assay to assess the cholinergic dysfunction which is one of the core pathologies of dementia and AD. Inflammatory cytokines like TNF-α, IL-6 was determined by ELISA method to evaluate the neuroinflammation which is aggravated by insulin resistance. C-reactive protein, a marker of neuroinflammation and neurodegeneration was also determined by ELISA. Mitochondrial dysfunction was evaluated estimating mitochondrial enzyme complex-I, II, III, IV depicting picture of viable and non-viable neuronal cellsHistopathology was done by H&E staining to find out apoptotic cells, neuroinflammation, and neurodegeneration. Molecular technique like RT-PCR for IRS-1, PI3-K, AKT, GSK 3-β and BDNF was performed for gene expression analysis.

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Potential role of Serpina3n in Neuroprotection after Ischemic Stroke

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Objective: Ischemic stroke is a ruinous CNS insult with finite effective therapeutical approaches. Primary limitation to develop novel restorative drugs is partially understood multidimensional underlying mechanisms and complicated crosstalk between them aiding to this pathophysiology. Recently, extracellular liberation of Granzyme B has been reported as an aider to neurodegeneration followed by CNS Ischemia. Inhibition of Granzyme B can be considered as one of the potential approach to reduce the neuronal damage. Murine serine protease inhibitor Serpina3n has a unique affinity for granzyme B while its human homolog serpina3 is incapable to inhibit granzyme B. By considering such reports, serpina3n can be considered as a potent therapeutical strategy to induce neuroprotection subsequent to ischemia by inhibiting granzyme B and serpina3 can be taken in parallel to investigate the underlying mechanism and unveil the contributing factors.

Methods: Recombinant serpina3n and serpina3 proteins were overexpressed using mammalian cells and purified. Recombinant proteins were tested for their invitro granzyme B inhibition. Neuroprotective effects of both proteins at different concentrations 20mM, 50mM and 100mM were investigated with N2A cell line by using invitro enzymatic ischemia/reperfusion model. Furthermore invivo experiments were undertaken by generating photothrombic ischemic mouse model and treated intranasally with 50mg/kg of recombinant proteins. Harvested brains were used to evaluate the infarct volume, immunohistochemical tests and western blot analysis for treated and untreated groups.

Key findings: Treatment with recombinant serpina3n and serpina3 increased cell viability in dosage dependent manner invitro followed by enzymatic ischemia. Post-ischemia 50mg/kg administrated intranasally induced neuroprotection confirmed by infarction volume. Exogenous proteins were successfully delivered to the ischemic lesion site across the blood brain-barrier.

Significance: Post- ischemia Serpina3n and Serpina3 induce neuroprotection and can be delivered successfully to the lesion focal area.

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Evaluation of a new therapeutic Sacroiliac Joint Manipulation Technique for patients with Sacroiliac Joint Dysfunction

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Purpose: The sacroiliac joint (SIJ) can be a cause of low back pain and sciatica. The aim of this study was to assess and introduce a new physiotherapy manipulation intervention for SIJ-related back and leg pain.

Methods: As a double-blinded, randomized trial, we evaluated the short-term therapeutic efficacy of a new intervention therapy against the current traditional practice. In this study, 60 patients with SIJ-related leg and back pain were studied. Following the initial assessment of each patient with respect to their perception of pain and quality of life (QOL), the effect of the treatment was evaluated. Using the visual analogue scale (annexure 1), perception of pain was assessed before and after the 1st, 2nd and 5th day (3rdday) of treatment. The instrument used for evaluation of quality of life was SF-36 (annexure1). At the outset of the study, QOL status was determined using this instrument. After the course of treatment by the assigned treatment method, each patient was reassessed using the SF-36 at the 5th week following a lapse of 4-week treatment free period. Except for having to drop out one patient for his failure to keep to the appointment, another 59 patients were treated successfully.

The results: The results show that the effect is highly significant at $\alpha = 0.001$(CI 4.3+0.7) for pain reduction and $\alpha = 0.001$(CI 1447.5+24.2) for QOL improvement. Of the 59 patients, 100% (n=30) have recovered from pain with the new treatment after the 3rd visit, while the recovery rate among patients who received the traditional treatment is 6.8% (n=29) after the 3rd visit.

Conclusion: In this double-blinded intervention study, the new manual therapy technique appeared to be the choice of treatment and more beneficial to the patients with back pain due to sacroiliac dysfunction than the traditional treatment. Further study to investigate its benefits with larger population should be encouraged.

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Advances in the diagnosis and treatment of Optic Neuropathies

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The aim of this presentation is to discuss the diagnosis, treatment and follow- up of different optic neuropathies. Meanwhile images from the new technology devices like OCT and the conventional machines like Perimetry will be shown. Also MR images of the orbit and brain and images of optic nerve problems will be the subjects of the main topic. There are some new developments in the treatment of the optic neuropathies and different ideas about novel treatments will be shared.

Optic Neuropathies: Optic neuritis (demyelination of the optic nerve): Acute, painful vision loss. Occurs mainly in women within the age range of 18- 45. Responds well to high dose intravenous steroids. Approximately 1/3 of the cases are seen with disc edema, 2/3 are retrobulber. Retrobulber cases progress to multiple sclerosis (MS) more frequently. The possibility of a woman patient to develop MS after isolated optic neuritis is about % 70 in ten years. The progression of optic neuritis to MS can be detected by OCT. Anterior ischemic optic neuropathy (non- arteritic): Acute, painless visual loss due to a stroke on the optic nerve head. Disc edema is present. Patients usually have hypertension or diabetes. Visual field defect is altitudinal. Cup to disc ratio is small. No definite treatment. Intravitreal injections (triamcinolone and anti- VEGF) may be tried in acute cases. Traumatic optic neuropathy: Occurs after a direct or indirect trauma to the optic nerve. Steroids are not recommended if there is head concussion. Surgery of the optic canal to decompress the nerve may be tried in early stages. There are reports about success with intravenous erythropoietin. Toxic optic neuropathy: Central, bilateral visual loss. There is a new report about improvement after methanol toxicity with the combination of erythropoietin and steroids. Radiation optic neuropathy: Bilateral visual loss months or years after radiation therapy of a brain tumor. There are new reports of improvement of vision with intravenous bevacizumab tharapy. Leber' s optic neuropathy: Painless, bilateral visual loss with central scotomas. Inherited by the maternal mitochondrial DNA mutations: m.11778, m.14484, m.3460. Idebenone treatment in early stage Leber's disease has been shown to be beneficial. Gene therapy is another option. Chronic relapsing inflammatory optic neuropathy: Steroid sensitive optic neuropathy which recurs after steroid withdrawal. Long term steroids or other immunosuppressive agents are used. There are other optic neuropathies with inflammatory, infectious, etc. etiology which can be discussed, too.

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