NEUROLOGY WORLD CONFERENCE

September 15-16, 2023

Theme
Latest Breakthrough in Neurology and Neuroscience

Hybrid (In-Person & Virtual)

Venue:
Hilton Garden Inn Miami Airport West, Miami, Florida, USA

EXHIBITOR

Advanced Infusion Care

SPONSOR

Capstone Vital Care Infusion Services

NWC 2023

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About
Precision Global Conferences

Precision Global Conferences is a highly established scientific conference organizer. We take high integrity in conveying your achievements to the world and emphasize your incredible work and scientific contribution. Precision global conferences have developed the progression, broadcast, persistence, research, and development activities in cancer, neurology, and nursing science,

We support the beacon of quality research works and efforts of academicians, researchers, scientists, doctors, and all the future young to be experts to confide their outstanding works fearlessly. Our primary goal is to make health care accessible and understandable to people. We are ecstatic to pass on the ray of research, developments, and cutting-edge therapies worldwide. Hence, we are here to organize and conduct highly esteemed conferences.

This conference will emphasize the outstanding works and their medicinal consequences through hybrid presentations. If you're searching for a perfect podium that can reflect your professional ethics and voice your appointment, we are here with the best team, welcoming your honourable presence.

About NWC

Neurology World Conference focuses on the crucial progression of innovations and breakthroughs through cutting-edge neurology research and practice. We aim to explore the new streamlines in the field and bridge neuronal Shortfalls in the industry. Neurology World Conference focuses on the crucial progression of innovations and breakthroughs through cutting-edge neurology research and practice. We aim to explore the new streamlines in the field and bridge neuronal Shortfalls in the industry.

The NWC 2023 agenda mainly focuses on bringing neurologists, neuro specialists, neuro physicians, academicians, medical professionals, and industry specialists from all disciplines to join us. We invite you to share the most recent findings with your colleagues. The upgrading technology and fast-paced lifestyles are diluting the mental health of people these days, giving rise to many such neurological diseases. Over the past 25 years, there has been an increase in neurological illnesses that cause illness, death, or long-term disability worldwide. Because of these problems, mortality rates are rising daily among people worldwide.

We are eager to expand our networks to honour learning and effort, and we are here to promote intellectual development and provide opportunities for networking and collaboration. The presentations will cover the most recent research and advancements in neurology. The session will be educational and feature interactions with peers and subject-matter experts, with a splash of inspiration.

We'll see that the best organizing team shares your best work on the best platform. We'll work hard to make the conference successful with the most significant planning schedules. It would be an incredible experience for you, us, and everyone attending the conference.

SPEAKERS

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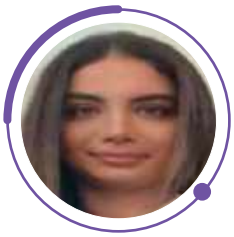
SPEAKERS



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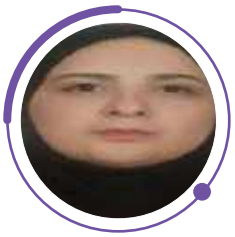

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SPONSPOR

Capstone Vital Care Immunoglobulin Therapy Services

Capstone Vital Care Infusion Services provides comprehensive immunoglobulin services to patients in the comfort of their own homes or at an alternate site. Our team of experienced and

knowledgeable infusion experts work with the provider to customize a care plan designed to optimize patient outcomes, reduce costs, and increase patient and provider satisfaction.

As one of the largest providers of Ig in the country, we offer:

- Access to multiple brands through direct manufacturer relationships and limited distribution agreements
- National contracts with Aetna, Cigna, Humana, and United Healthcare
- Access to regional and local contracts Capstone Vital Care is committed to providing quality.
- Care with a comprehensive and high-touch approach:
- A seamless referral process allows patients to start treatment quickly.
- Customized care plan
- Patient and caregiver education regarding therapy, techniques, and supplies
- In-home or alternate site care option
- 24/7 on-call support from experienced pharmacists and nurses

Clinical Excellence

- Highly trained infusion experts
- Intravenous or subcutaneous routes of administration (IVIg or SCIg)
- Clinical monitoring by Ig-trained nurses and pharmacists
- The Proactive order refill process

EXHIBITOR

Advanced Infusion Care

At Advanced Infusion Care, we apply a team approach to intravenous (IV) and subcutaneous (SubQ) immunoglobulin therapy patient care. Collaboration between physicians and the nationally accredited AIC home infusion team ensures the consistent delivery of patient-specific, specialized in-home infusion services to patients across the country, helping to improve outcomes—and lives.

We have more than 100 fully certified and highly trained nurses on staff, all of whom are committed 1 to doing more to provide the best possible care for patients. On-staff nurses provide care to 97% of AIC patients.

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DAY 1

KEYNOTE SPEAKERS

Expressive Arts Tools for Neurologists to Combat Burnout

Juliana Fort*, MD, MPH, MBA, Karine Scheuermaier
(Psychiatry Department, LSU Health Shreveport, Shreveport, LA, USA, Eleanore Knox, MD (LSUHS, Shreveport, LA, USA
*Clinical Associate Professor in the Department of Psychiatry and Behavioural Medicine at Louisiana State University Health Shreveport, USA

Six out of ten neurologists in the United States are experiencing some form of burnout, and research suggests similar rates worldwide. Burnout is characterized by the ICD 11 as "overwhelming fatigue and emotional exhaustion, feelings of cynicism, detachment from the job, a sense of ineffectiveness, and a lack of personal accomplishment". The cumulative stress of caring for a person with a chronic neurological disorder can contribute to burnout, both among physicians and patient families. Expressive arts techniques using visual art, drama, music, creative writing, and movement, can be used to promote neurologists' resilience and self-care.

Biography

Dr. Juliana Fort is a Clinical Associate Professor in the Department of Psychiatry and Behavioural Medicine at LSU Health Shreveport where she is the Psychiatry Medical Student Clerkship Director and the Associate Director of the Child and Adolescent Psychiatry Fellowship. Dr. Fort is a graduate of the LSU Health Shreveport -- School of Medicine and completed her Psychiatric residency and Child and Adolescent Fellowship in the Department of Psychiatry at Tulane Medical School in New Orleans, Louisiana. She is board-certified in Child and Adolescent, Geriatric, Forensic, and Addiction Psychiatry and is a distinguished fellow of the American Psychiatric Association. She is a registered play therapist/supervisor and enjoys training in the Expressive Art Therapies that enrich psychotherapy, wellness, and personal growth through Drama Therapy, Improvisation, and Art and Poetry therapy.

Effect of Thymosin β4 on Lipopolysaccharide-Stimulated Brain Microvascular Endothelial Cells Remodeling: A Possible Role in Blood-brain Barrier Injury

Sudhiranjan Gupta

Biomarkers & Genetics Core, VISN 17 Centre of Excellence for Research on Returning War Veterans, USA

War Veterans are particularly more prone to mental illnesses due to their prior encounters with multiple traumatic brain injuries (TBI) while serving on active duty, specifically in war zone areas. TBI is known to cause mortality or serious neurological disabilities among survivors. TBI has been previously reported to elicit several pathological processes, including neuroinflammation and blood-brain barrier (BBB) disruption, leading to secondary brain damage and the subsequent impairment of the neurovascular unit. Although several drugs have been shown to exhibit promising effects for TBI, the repertoire of currently available therapeutic strategies remains limited. Thymosin 4 (Tβ4) is a 43-amino acid G-acting sequestering peptide that has been previously documented to confer neuroprotective potential in TBI models. However, its role in BBB function remains unclear.

Further research into the mechanism of BBB disruption induced by TBI and its' specific role in neurovascular pathophysiology is necessary. In the present study, the protective effects of Tβ4 in lipopolysaccharide (LPS)-stimulated gene expression of several tight junction proteins, inflammatory genes, apoptotic genes, and adhesion genes in human brain microvascular endothelial cells (hBMVECs), one of the pivotal cell types in the BBB, were reported. These data suggest that pre-treatment with Tβ4 reversed the LPS-induced damage of BBB components in hBMVECs. Furthermore, these results identified neuregulin 1 to be a possible target for Tβ4. Therefore, it can be proposed that Tβ4-mediated cellular signalling in hBMVEC may be vital for understanding the association between the BBB and TBI pathophysiology, which warrants further investigation.

Biography

The presenter did MS and Ph.D. in Molecular Biology. The presenter completed his post-doctoral training at Cleveland Clinic in molecular cardiology and worked as a Scientist for three years before moving to Texas A&M University as a tenure-track faculty in the Department of Internal Medicine. The presenter ran a vibrant lab for 9 years there and trained several students and post-doctoral fellows. The presenter published many scientific articles in peer-reviewed journals. The presenter had an excellent track record of achieving many national-level research grants in cardiac research. The presenter moved to the VISN17 Centre of Excellence, Waco, TX, in 2019 to study neurological diseases/ disorders, primarily focussing on the molecular mechanism of blood-brain barrier dysfunction in traumatic brain injury. The presenter is also working on PTSD and engaging in identifying biomarkers for PTSD.

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DAY 1

ORAL SPEAKERS

Cholesterol Management in neurology: Balancing Beneficial Cardiovascular Effects versus long-term Cognitive side-effects of statins

Jurcau Anamaria*, Jurcau Maria Carolina[1]
[1]Department of Psycho-Neurosciences and Rehabilitation, University of Oradea, Oradea, Romania *Senior neurologist at the Clinical Emergency County Hospital Bihor, Associate Professor at the Faculty of Medicine and Pharmacy, University of Oradea, Department of Psycho-Neurosciences and Rehabilitation, Romania

The results of the Scandinavian Simvastatin Survival Study (4S) released in 1994 led to a series of studies that showed the beneficial effects of statins (HMG-CoA reductase inhibitors) in preventing vascular events. Based on these results, successively issued guidelines pushed for lower and lower levels of LDL-cholesterol as targets for statin treatment. However, post-marketing reports that drew attention to transient cognitive impairment and short-term memory losses caused by statin treatment prompted the Food and Drug Administration (FDA) to issue a warning regarding the potential for reversible cognitive impairment in statin users in 2012.

The cholesterol content of the nervous system is very high, cholesterol being the basic constituent of membranes and myelin sheaths and influencing the properties of membranes as well as the activity of receptors and ion channels. Its synthesis via the mevalonate pathway is crucial in the early stages of development, but the transcripts of the enzymes necessary for cholesterol synthesis remain in neurons throughout life and serve to obtain dolichols, ubiquinones, and isoprenoids that modify proteins, and small GTPases which are critical for axon growth and guidance, growth cone motility, dendritic arborization, as well as synapse formation. Although the brain is largely separated from the periphery through the blood-brain barrier, most of the currently marketed statins are lipophilic and can gain access to the CNS, where they can inhibit HMG-CoA reductase in the mevalonate pathway, thereby interfering with the myelination process and with synaptogenesis, with the formation of neuronal circuits, as well as with neuronal excitability via disturbing the function of ion channels. In addition, coenzyme Q10 is depleted, leading to mitochondrial dysfunction, which has been convincingly implicated in the pathogenesis of dementia.

As neurologists, we use statins mainly in primary or secondary prevention of ischemic strokes. Nonetheless, there are many subtypes of ischemic stroke. While in thrombotic stroke LDL-cholesterol and atherosclerotic plaques have a significant contribution, in other subtypes, such as embolic stroke, the role of cholesterol is less well established. Moreover, cardioembolisms tend to occur in older patients, often frail and in poor nutritional status, with lower LDL-cholesterol levels compared to patients with thrombotic events. We would prevent strokes in our patients, but we would also avoid trading the brain for the heart.

In 2013, the American College of Cardiology/American Heart Association reviewed the safety issues raised by statin therapy and concluded that the evidence on statin-induced cognitive impairment is inconsistent. However, their conclusions were based mainly on the JUPITER, PROSPER, and HPS trials, which did not perform detailed neuropsychological assessments because cognitive dysfunction was neither a primary nor secondary outcome measure. Several studies addressed the issue of the effects of long-term use of statins on cognition and yielded conflicting results. A meta-analysis of observational studies (Poly et al., 2020) concluded that hydrophilic statins were associated with a lower risk of all-cause dementia, while lipophilic ones lowered the risk of AD but not of vascular dementia.

Until future studies provide a definite answer to the question of the effect of long-term statin use on cognition, we suggest thoroughly balancing the benefits versus possible risks in elderly patients before prescribing statin therapy, favour hydrophilic over lipophilic ones, and using ezetimibe or PCSK9 inhibitors to meet the target LDL-cholesterol levels.

Biography

Jurcau Anamaria: MD, PhD, Senior Neurologist at the Clinical Emergency County Hospital Bihor, Associate Professor at the Faculty of Medicine and Pharmacy, University of Oradea, Department of Psycho-Neurosciences and Rehabilitation, Romania. Main areas of interest: ischemic stroke; oxidative stress; neurodegenerative diseases. She published several manuscripts highlighting the involvement of oxidative stress and mitochondrial dysfunction in the development of ischemia/reperfusion injuries as well as in the molecular pathophysiological pathways of neurodegenerative diseases such as Alzheimer's disease and Huntington's disease. In close connection with these topics, I was also interested in the relationship between cholesterol and cognitive functions in elderly patients.

Accelerated Dysregulation of Immune and Neuronal Markers in HIV-1 Infected and Aged Humanized Mice

Prasanta K Dash, Chen Zhang, Hang Su, Aditya Bade, Larisa Poluektova, Santhi Gorantla, Howard E Gendelman
Assistant Professor, Department of Pharmacology and Experimental Neuroscience, University of Nebraska Medical Center

Despite virologic suppression on ART, people living with HIV (PLWH) are at high risk of age-related diseases and accelerated aging. However, the underlying mechanisms contributing to this increased risk have not been completely defined but may include genomic and epigenetic changes, cellular senescence, mitochondrial dysfunction, and metabolic alterations. We utilized CD34-NSG humanized mice, which supports long-term systemic engraftments of a functional human immune system to investigate aging-associated signalling pathways in late-stage HIV-1 infection. CD34-NSG humanized mice were infected with HIV-1ADA at 20 weeks of age following humanization and maintained for an additional 40 weeks. Immune and viral profiles were analysed at 0, 4-, 8-, 16- and 40 weeks post-infection. At 60 weeks of age, mice were sacrificed, and brains were isolated to evaluate the viral expression and key aging markers utilizing RT qPCR, immunofluorescence, and transcriptomic assays. A gradual decline of the CD4+ T cells and steady viral replication were observed in infected aged mice at the endpoint. Transcriptomic analysis of infected aged brains demonstrated an overall downregulation of the genes involved in human aging. The top 4 differentially expressed genes are COL1A, ELP3, LMNA, and SIRT1. Ingenuity pathway analysis further revealed several major pathways affected in HIV-infected aged mice were related to neutrophil activity, pyroptosis, phagosome formation, and Granzyme A and LXR/RXR signalling. The transcriptomics data corroborated with the immunofluorescence data for neuronal and non-neuronal markers in several brain compartments.

To summarize, CD34-NSG humanized mice can be used as a model system to study HIV-associated aging. Upregulation of immune-senescence-associated and downregulation of major signalling pathways was observed in HIV-1 infected aged mice. To explore the underlying mechanisms that may contribute to the increased prevalence of age-related comorbidities in PLWH under ART treatment, it is necessary to identify HIV-aging-related biomarkers, which will help to design appropriate intervention strategies for clinical translation.

Biography

I am an Assistant Professor in the Department of Pharmacology and Experimental Neuroscience at the University of Nebraska Medical Center, Omaha, NE, USA. I did my Ph.D. in India, working on Epidemiology and NeuroAIDS of HIV-1 infection and Molecular Characterization of viral clones from an HIV-1 demented Indian Patient. My current focus is to look at the molecular details associated with HIV and aging and find new biomarkers using a suitable small animal model of the HIV disease by employing NSG-humanized mice. My other research focuses on drug-drug interactions and molecular pathways for HIV therapeutics and elimination. My work led to the "first" eradication of HIV-1 infection in humanized mice using sequential combinations of long-acting slow effective, release antiretroviral therapy and CRISPR-Cas9. In addition, my laboratory works on evaluating viral compartmentalization through analyses of tissue viral reservoirs under highly suppressive antiretroviral HIV-1 therapies with a special focus on the brain. We have recently developed a highly sensitive in vivo mice viral outgrowth assay using humanized mice as donors and recipients to capture replication-competent latent HIV-1 from peripheral, myeloid, and central nervous system (CNS) reservoir compartments, which will be an important tool to evaluate HIV-cure therapies. My work also includes studies of the interrelationships between tissue histology and quantitative measurements of the virus production and examinations of virus-associated biomarkers through metabolic profiling in small animal models of human disease. My background is in molecular retrovirology, pharmacodynamics, immunology, and neuroscience experience with more than 40 publications in high-profile journals, including corresponding author publications and invited editorials in respected journals. Our work to look at the distribution of HIV in the CNS compartment in humanized mice is funded by NIMH. I have trained more than ten summer students, and five research technologists, co-mentored one graduate student, and currently have one Ph.D. student. I have received consecutive Young Investigator Awards from CROI and SNIP societies from 2011 to 2015. In 2020 I received the New Investigator Award from the University of Nebraska Medical Center.

EEG Phase Amplitude Coupling as New Biomarker of Cortical Dysfunctions in Amyotrophic Lateral Sclerosis

Veronique Marchand Pauvert
Laboratory of Biomedical Imaging, Sorbonne University/Inserm, France

Amyotrophic lateral sclerosis (ALS) is characterized by the degeneration of upper (brain cortex) and lower (motoneurons) motor neurons (respectively, UMN & LMN), but its diagnosis mostly relies on LMN affection, common to other diseases. Besides delaying treatment, this also limits clinical trials by postponing patient inclusion, and the gold standard is still LMN evaluation primarily (using EMG), excluding UMN assessment for testing therapy efficacy. Thus, there is a crucial need for UMN biomarkers based on a reliable and easy-to-use approach. Methods relying on transcranial magnetic stimulation (TMS) and magnetic resonance imaging (MRI) revealed early cortical dysfunction, even at the presymptomatic phase of familial forms, that does not mirror specific LMN diseases. In both human and mouse ALS models, cortical dysfunction precedes LMN dysfunction and negatively correlates with survival. Cortical dysfunction is not specific to ALS, but its association with LMN signs would help to ensure diagnosis and prognosis. However, TMS and MRI have limited application in the clinical routine of ALS for physiological and technical reasons. Electroencephalogram (EEG) has the potential to fulfil the unmet need for quantitative and reliable biomarkers of cortical dysfunction thanks to recent methodological advances. Our objective was thus to investigate PAC in preclinical models of ALS and in patients to determine whether it could be used as a new biomarker of cortical dysfunctions in ALS. In my talk, I will present the results of the Strasbourg team led by C. Rouaux, who has evidenced the link between PAC and cortical excitability and that PAC is reduced in ALS mice. I will detail the results we obtained in my group in i) control subjects in whom we demonstrated that PAC is modified when the brain cortex is modulated by repetitive TMS and ii) in ALS patients in whom we revealed a specific decrease of PAC in the primary sensorimotor cortex, as observed in preclinical models.

Biography

Pr. Marchand-Pauvert (Ph.D.) is the Research Director at Inserm (National Institute for Health and Medical Research), an equivalent position to a University Professor. She is a neurophysiologist with specific expertise in the sensorimotor system. She is mainly involved in studying the corticospinal connectivity underlying voluntary movements in humans. She is particularly well-known for her great knowledge of the human spinal circuitries. Since 2014, she has led the neuroscience team in the Laboratory of Biomedical Imaging at the Pitié-Salpêtrière Hospital, where she develops research programs coupling neuroimaging and electrophysiology to better understand the physiology of the human sensorimotor system, the ALS pathophysiology and to identify biomarkers.

A Case Study: Concussion May Result in New-onset Bipolar Disorder

Kent Sabatose*, DC, DIBCN, Dr. Murat Ibatullin, MD, PhD, Nichole Cufino,
BS Lake Erie College of Osteopathic Medicine, Bradenton, Florida, United States
*Lake Erie College of Osteopathic Medicine, USA

Introduction: Emotional dysregulation following a concussion is well-established in the literature. The new onset of major psychiatric diseases such as bipolar disorder (BPD) post-concussion has not been investigated. BPD typically presents with an initial depressive episode followed by mania and concurrent depressive and manic states. Multiple theories explaining the etiology of BPD exist, including the diathesis-stress model (DiSM), though an accepted theory is not established.

Case Presentation: A 50-year-old ambidextrous male with comorbid ADHD inattentive type, OCD, and a family history of BPD, suffered a motor vehicle collision (MVC) resulting in a grade II concussion. Brain MRI 2 months post-MVC showed negative susceptibility weighted imaging (SWI), increased fluid-attenuated inversion recovery (FLAIR) signal in the frontal and parietal lobes, and abnormal diffusion tensor imaging (DTI) with cerebral fractional anisotropy score in trauma (C-FAST) the score of 4. New onset BPD was diagnosed, leading to unemployment and homelessness followed by a cascade of medical crises including ascending aortic aneurysm rupture and stroke. MRI 6 months later revealed an increased FLAIR signal in the right frontal, parietal, and occipital lobes with frontal encephalomalacia. Scattered SWI blooming clustering in areas of increased FLAIR signal. Postconcussion syndrome symptoms as well as BPD persisted unchanged indefinitely responding to Zyprexa and Trileptal pharmacotherapy.

Conclusion: This case study may serve as a real-world example of the DiSM in the etiology of BPD post-concussion and its application in clinical medicine.

Biography

Kent Sabatose graduated from Palmer College of Chiropractic receiving a diplomate in functional neurological rehabilitation. Before beginning medical school in 7/2022, he was the director of neurological rehabilitation at Florida Surgery Consultants. There he specialized in non-invasive neuromodulatory techniques including transcranial magnetic stimulation, direct current brain stimulation, hyperbaric oxygen therapy, and neurofeedback. He has successfully worked with patients suffering from conditions such as traumatic brain injury, stroke, Parkinson's, multiple sclerosis, and migraine utilizing non-invasive neurological rehabilitation strategies. He is an aspiring neurosurgeon and hopes to combine his passion for neurological rehabilitation with the field of neurosurgery.

Painful Neurotrophic Keratopathy

Leyla Yavuz Saricay[1, 2], Eric Moulton[1,3]
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[2]Department of Ophthalmology, Mass Eye and Ear Infirmary, Harvard Medical School, United States
[3]Brain and Eye Pain Imaging Lab, Pain and Affective Neuroscience Center, Department of Anaesthesia, Critical Care and Pain Medicine, Boston Children's Hospital, Harvard Medical School, MA, United States

Neurotrophic keratopathy (NK) is a degenerative corneal disease that is the result of impaired corneal innervation by the ophthalmic branch of the trigeminal nerve. Though NK is associated with decreased corneal mechanosensation, recent studies indicate that spontaneous pain can also occur in such patients. This presentation will review the state of the field regarding the relationship between symptoms of pain and the clinical ophthalmological measures in patients with NK, including an emphasis on in vivo corneal microscopy findings. NK is associated with decreased measures of corneal nerve density but also can demonstrate clinically significant pain along with increased dendritiform cell density, a marker of localized peripheral inflammation. Chronic ocular pain may develop with NK, with nerve damage and inflammation playing roles as contributing factors. Increased ocular pain with decreased corneal nerve fibers is suggestive of a sensitization and/or dysregulation of central nervous system circuits related to nociceptive processing.

Biography

Dr. Yavuz Saricay is a clinician scientist interested in research specifically in neurotrophic eye diseases. She is currently a clinical fellow (Fellow in Cornea, External Disease, and Refractive Surgery) at Mass Eye and Ear Infirmary and completed 2 years of paediatric ophthalmology fellowship and ophthalmic genetic clinical fellowship at Boston Children's Hospital. Her research interest in neurotrophic keratopathy (NK), NK management, as well as corneal nerve regeneration first started during her 2 years of research fellowship under the supervision of Pedram Hamrah, and she has continued to investigate and publish about the topic since then.

Continuous Epidural Analgesia: An Analgesic Alternative in Low Back Pain with Disabling Radiculopathy

Carla Retroz Marques
Anaesthesiology Consultant at Coimbra University Hospitals Centre, Portugal

A male patient, middle-aged and BMI of 25.7 kg/ m2 (weight 69 kg, height 1.64 m) was referred to the chronic pain unit 10 years ago for shoulder and residual thalamic pain following a cerebrovascular accident. This pain was treated with conventional analgesic therapy. Five years ago, the patient reported severe low back pain (LBP) with bilateral radiculopathy in the lower limbs without 'red flags. The medical history included diabetes, controlled hypertension, and coronary artery bypass graft surgery. At that moment, the patient was not taking any anticoagulation therapy. CT revealed moderate spinal stenosis in L3-L4 and root compression due to herniated disc protrusion in L4-L5. Despite six months of several systemic multimodal analgesic therapies (increasing doses and rotation of strong opioids/adjuvants by the WHO ladder), the LBP prevented the patient from physiotherapy, having a strong impact on daily living activities (DLA), mood, and willingness to live. The pain intensity (9-10 on the visual analogue scale) with bilateral radiculopathy (L4-L5 dermatomes distribution, Lasègue sign positive and without neurological deficits) had the characteristics of intractable pain through conventional treatment. An invasive analgesic technique was then considered with the patient's consent. The patient was informed of the protocol safety when the criteria are followed strictly and of the general and rare specific risks, namely dura-mater accidental puncture, subdural haematoma, urinary retention, and local/systemic infection. A protocol of continuous epidural technique with a tunneled catheter was then initiated. A diagnostic and therapeutic technique was used with a single-shot lumbar epidural for immediate and prolonged relief of severe bilateral pain and functional disability (6.25 mg of levobupivacaine, 14 mg of betamethasone dipropionate, and 1.5 mg of morphine). After 2 weeks without pain, there was an LBP recurrence requiring the protocol's second stage to be followed. With the patient seated in his comfort position, a median interlaminar epidural approach was performed at L4-L5 intervertebral level, followed by 20G tunnelled catheter placement without complications. Simultaneously, with the gradual discontinuation of systemic opioids, the epidural perfusion analgesia was administered by sequential EIPs (capacity 65 mL for 5 days; 0.5 mL/ hour; with 13 mg/day of levobupivacaine 0.1% + 5 mg/day of morphine), providing pain relief without adverse effects, while ensuring the restoration of DLA.

An MRI revealed a migrated hernia fragment lodged in the L4 root without neurological compression. After ten weeks, the epidural catheter was removed without complications or inflammatory/infection signs (negative bacteriological analysis of catheter tip and blood culture). After reassessment by the neurosurgical team, this favourable evolution of symptoms excluded criteria for surgical intervention.

During the last four years, the patient did not experience lumbosciatic pain recurrence and restored DLA. The original thalamic pain has been controlled with mild analgesic therapy. This clinical case demonstrates that a technique widely used in other contexts can be adjusted for outpatient treatment of non-cancer pain as long as the safety criteria are strictly respected. Studies confirm that continuous epidurals with opioids, anaesthetics, and corticosteroids for LBP result in the control of disabling pain crises, reducing doses of systemic opioids with significant improvement in quality of life.

Biography

Carla Retroz Marques is an Anaesthesiology Consultant at Coimbra University Hospitals Centre. She completed her Graduation of Medicine at the Faculty of Medicine of the University of Coimbra (FMUC) (1985-1991). She earned her master's degree in Anaesthesiology and Pain Therapy from the University of Coimbra (FMUC) and her master's degree in Palliative Care from the Faculty of Medicine of the University of Coimbra (FMUC). She is a Member of the Chronic Pain Clinic of Coimbra University Hospitals Centre. She gives Guidance Training in Anaesthesiology and Pain Therapy.

Effect of Disc Dehydration on the Clinical Symptomology of Chronic Low Back Pain. Clinical Radio Study by Functional MRI

Mammari MDE
Military Hospital of Oran, Algeria

Background and aims: The objective of this study is to compare clinical data from cases of chronic low back pain with those from functional MRI by evaluating the effects of disc dehydration.

Material and method: This is a descriptive study covering 124 cases of chronic low back pain (young adults) from September 2021 to April 2022, benefiting from an evaluation comparing the clinic (VAS-pain, root irritation signs, chronic smoking, Oswestry Questionnaire) with functional imaging data (CDA values of NP and AF). The exploration of the results is carried out by the SPSS-20 software.

Results: The results of the functional MRI expressed by the values of the discal CDA of the Nucleus Pulposus (NP) and the Annulus Fibrosus (AF) showed a very significant correlation with the intensity of pain, with the Oswestry score for the NP. The results show that there is no evidence of a statistically significant link between signs of root irritation, as well as with chronic smoking.

Discussion-Conclusion: The study shows that PN dehydration has a significant link with chronic low back pain and disability. There is no correlation between the signs of root irritation and the proportion of water in the lumbar intervertebral disc. Similarly, the role of tobacco is not established. Other factors would come into play.

Biography

Mammari MDE is a Physical Doctor Military Hospital in Oran, Algeria. He worked as a Researcher in the Department of Physical Medicine and Rehabilitation at the University of Oran.

Economic and Health Impact of the First Pass Effect in Mechanical Thrombectomy for Acute Ischaemic Stroke Treatment in Spain

Eva Maria Gonzalez Diaz
Diagnostic Neuroradiology, University Hospital of Cruces, Spain

Objective: The clinical benefit of mechanical thrombectomy (MT) in acute ischemic stroke treatment, is correlated to the degree of reperfusion achieved. The First Pass Effect (FPE) is defined as complete/near revascularisation of the large-vessel occlusion [modified Thrombolysis in Cerebral Infarction (mTICI)2c-3] after a single device pass. FPE can potentially be one of the primary goals in the treatment of ischemic stroke due to large vessel occlusion (LVO)from a clinical and economic viewpoint.

Methods: The model simulates a hypothetical cohort of 1000 patients with clinic-demographics characteristics based on the STRATIS registry (Systematic Evaluation of Patients Treated with Neurothrombectomy Devices for Acute Ischaemic Stroke). STRATIS registry patients were classified into 2 groups: patients with a final mTICI ≥2b (used for the base case analysis), and patients with final mTICI (0–3) (used for the alternative scenario). Afterward, patients in both groups were stratified into FPE and non-FPE groups. The model had a two-phase structure, consisting of an acute-subacute phase from stroke onset to 90 days, and a rest-of-life phase 91 days after stroke to the end of the patient's life.

Results: Our results suggest that the FPE group had significantly better clinical outcomes at 90 days after stroke compared with the non-FPE group in the base case scenario (mRS 0–2: 66.2% vs 54.6%, $p<0.005$) and in the alternative scenario (mRS 0–2: 66.9% vs 50.6%, $p<0.0001$). In the base case scenario, the model estimates an average lifetime cost per patient of €97,206 for the FPE group and €113,790 for the non-FPE group. Overall, the FPE group generated a cost reduction of €16,583 per patient in a lifetime horizon. Cost reductions are predicted to be greater when nursing/residential care costs are included, leading to a savings of €30,072 per patient. The alternative scenario results in a cost reduction per patient of €21,910 estimated and a further reduction of €44,289 when nursing/residential costs are considered. In terms of health outcomes, achieving FPE results in an incremental QALY gain of 1.2 and 1.75 in the alternative scenario.

Conclusion: Acute ischemic stroke patients treated with neurothrombectomy devices and who achieved FPE after MT showed significantly better functional clinical outcomes and were associated with important healthcare cost savings per patient compared with those who did not achieve FPE.

Biography

Eva María González Díaz. I was born in 1973 in Bilbao, Spain, where I currently live. I graduated in 1997 from the University of the Basque Country and after passing the entrance exam for the choice of the specialty I opted for radiology, given my interest in interventional radiology. After completing my four-year training as a resident internal radiology doctor at the University Hospital of Cruces, Bilbao, in 2002, I began my information as an interventional neuroradiologist. During the first two years, I worked as a diagnostic radiologist as well as an interventionist, later given the requirement in my center to promote interventional neuroradiology, it was necessary to dedicate myself exclusively to it. It was then that in 2003 I had face-to-face training at the Radclyffe Infirmary Hospital in Oxford United Kingdom. Subsequently, I carried out several pieces of training in German, French, and Turkish hospitals where the activity of interventional neuroradiology was highly developed. During this time, interventional neurology had an important technological advance in the treatment of aneurysms and brain malformations.

In 2009, during my pregnancy, I devoted myself to diagnostic neuroradiology and it was after my maternal leave that I became the head of the department. In 2012, we started with the 24-hour guards of Interventional Neuroradiology, to perform the endovascular treatment of both brain aneurysms and the treatment of ischemic stroke through mechanical thrombectomy. This treatment was an important advance for the clinical evolution of stroke, but it meant an important change in our lives for 15 days of call per month were required. As for the scientific part, I have participated as a speaker in several congresses and scientific meetings both nationally and internationally.

I am the author of several articles referring to interventional neuroradiology in national and international journals and the lead author of the recently published article in the British Medical Journal. I also participate as a professor in both practical and theoretical training in interventional neuroradiology at the University of Santiago de Compostela Galicia Spain. I am a member of the committee on vascular tumors and neurovascular pathology of the University Hospital of Cruces, and I belong to scientific societies such as the Spanish Society of Neuroradiology and the Spanish group of interventional neuroradiology.

Masgutova Neurosensorimotor Reflex Integration (MNRI®) – A State-of-the-Art Primary Infant Reflex-Based Approach for Facilitation of Human Neurodevelopment

Isabelle Renard Fontaine
Paediatric Physical Therapist, MNRI® Core Specialist and Instructor, USA

The MNRI® Method, created by Dr. Svetlana Masgutova, post-PhD, a world-renowned expert on reflex integration, consists of a non-invasive manual Neuromodulation technology. It uniquely recognizes the Dynamic Process of Maturation specific to each infant's reflexes and uses these reflex circuits - "readily given" genetic codes- to facilitate Neurodevelopment. It has been called a "Missing Link" in neurorehabilitation by an impressive array of professionals for its high therapeutic effect. The MNRI® Program has broad application, reaching an estimated 300,000 children and 80,000 adults in over 40 countries. Dr. Masgutova developed the unique and statistically verified MNRI® Reflex Assessment, which evaluates the function of primary reflex patterns based on specific parameters and multiple factors. The MNRI® Reflex Assessment provides a clear understanding of the level of reflex maturity and the overall state of the sensory-motor system in an individual. The MRNI metrics tool allows for identifying normal, neurophysiological mature, or abnormal reflex patterns expressed at any age -in newborns, infants, children, and adults-. Dr. Masgutova's research evidences the correlation between pathological, dysfunctional reflex patterns and medical diagnoses. The lack of reflex development and the disruption of reflex dynamics (i.e., a blockage at the primitive form of the reflex, stress, and trauma) consistently compromises the neurodevelopment causing delays, deficits, and maladaptive effects from mild to extreme severity. The goal of MNRI® programs working with Neurodevelopmental disorders is to use reflex patterns neuromodulation to regulate excitation-inhibition of the CNS and increase stress resilience and neuroplasticity. Examples of neurodevelopmental disorders include autism spectrum disorder, cerebral palsy, Down syndrome, genetic disorders, speech/language and social deficits, specific learning disability (SLD), trauma and PTSD, and behavioural/emotional dysregulation. More than three decades of practical work and IRB-approved scientific research and publication demonstrate the significant positive effects of the MNRI® on sensory-motor integration, neurophysiological and physical, behavioural, and emotional, language, cognitive, and social development among a large variety of paediatric and adult population suffering from diverse neurodevelopmental and health conditions. This lecture offers a brief overview of the concepts and foundational principles of MNRI® and discusses the unique value of MNRI® Reflex Assessment through case demonstrations and MNRI® scientific research.

Biography

Isabelle has acquired more than 28 years of experience as a Physical Therapist, predominantly with premature babies, newborns, infants, and children. Isabelle is an MNRI® Core Specialist and Instructor with extensive expertise on the effect of the MNRI® as an early intervention tool for babies and toddlers with neurodevelopmental challenges. She has accrued more than 2,000 clinical and practical hours working directly with Dr. Svetlana Masgutova and her international team during international MNRI® Family Educational Conferences and Clinics. A lecturer and subject matter expert for The Masgutova Graduate School of Neuro Developmental Sciences, Isabelle has developed the curriculum for and instructs the Neuromodulation for Infants and Toddlers university-level course and serves as an instructor for the Primary Movement and Biomechanics Integration and Neuromodulation and Stress Hormone Reflex Neuromodulation courses. In addition, Isabelle is co-coordinator of the MNRI® Reflex Screening in Infants Development Program for Paediatricians and Physicians. She also regularly conducts lectures on the latest advancements and applications in MNRI® and neurodevelopment through the Svetlana Masgutova Educational Institute's MINDS Group. Isabelle's professional and personal passion is helping premature babies, infants, and children with life-survival challenges reach their highest level of neurodevelopment.

Increasing the Therapeutic Potential of BBB Penetrating Drugs via Alteration of Parenchymal/Cerebral Spinal Fluid Clearance Rate

Benjamin J Umlauf
Assistant Professor, Department of Neurosurgery, Dell Medical School, USA

Accumulating physiologically relevant amounts of drugs within the central nervous system (CNS) remains a major hurdle in treating many encephalopathies. For example, temozolomide (TMZ) chemotherapy is administered at the maximum tolerated dose to patients with glioblastoma (GBM) but imparts only a few months of survival benefit. To improve TMZ efficacy via enhanced CNS accumulation, we developed a method to reduce the clearance rate of TMZ from the brain. Recent glymphatic studies demonstrate the interconnectivity between the fluid in the brain's parenchymal and ventricular spaces. We reasoned that enhancing the accumulation of a drug in the cerebral spinal fluid (CSF) of the ventricular space will also improve the accumulation of the drug in the parenchyma, where many encephalopathies, including GBM, present. We altered the biophysical properties of motile ependymal cilia to improve the accumulation of a blood-brain barrier penetrating therapeutic in the CSF. Ependymal cells coat the ventricles and display motile cilia that mechanically propel CSF through the ventricular space. We propose impairing ciliary motion slows CSF turnover without affecting the overall amount of CSF in the CNS. We identified FDA-approved therapeutics known to impair airway cilia as a "side effect" and demonstrated that these drugs also impair ependymal cilia. A panel of cilia inhibitors was administered into the ventricle of mice bearing orthotopic GBM in combination with systemic temozolomide (TMZ). Five of the seven cilia-inhibiting drugs significantly improved overall survival when combined with TMZ compared to controls. The lead candidate, lidocaine, demonstrated a synergistic relationship with TMZ, and 100% of animals treated with the combination regimen survived to the study endpoint tumour-free. Combining intraventricular lidocaine with TMZ is well tolerated and results in a 40-fold increase in brain temozolomide levels throughout the study compared to controls. Finally, we observe 100% tumour-free survival after treating mice bearing a human PDX orthotopic xenograft GBM with the combination of lidocaine and TMZ and 40% tumour-free survival when treating mice bearing an un-methylated MGMT human PDX orthotopic xenograft GBM that is resistant to TMZ treatment alone. These studies offer a new concept for treating malignant brain tumors and a potential scheme to improve the treatment of other encephalopathies via enhanced brain accumulation of CNS penetrating drugs.

Biography

Benjamin J. Umlauf, Ph.D., is an assistant professor in the Department of Neurosurgery at Dell Medical School. His research is focused on developing solutions for unmet clinical needs using immunological, biological, and chemical techniques. Significant foci include the development of novel therapies for treating solid tumors, identification of targeting ligands using multiple platforms including viral, yeast, and bacterial systems, a site-specific combination of biological and synthetic chemistry products, and enhancing the accumulation of blood-brain barrier penetrating drugs within the central nervous system.

Dr. Umlauf began his research career at the Mayo Clinic in Rochester, MN, then attended graduate school at UT Southwestern Medical Center in Dallas, TX, where he received a Ph.D. in cellular and molecular biology. He next went to the University of Wisconsin-Madison for his postdoctoral fellowship in the departments of Chemical and Biological Engineering and Neurosurgery.

His previous work includes identifying lung cancer-targeting ligands with defined subcellular trafficking patterns, developing novel peptide-based immunotherapy platforms, and discovering methods to target encephalopathies via blood-brain barrier disruption rather than disease-associated receptors.

Age- and Pathology-related Transcriptomic Changes and Treg function in APP/PS1 Transgenic Mouse Model for Alzheimer's Disease

Mai Mostafa*, Mai M. Abdelmoaty, Pravin Yeapuri[2], Jatin Machhi[13], Yaman Lu[1], Krista L. Namminga[1], Rana Kadry[2], L. R. Lee Mosley[1], and Howard E. Gendelman[1]

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*Postdoctoral Research Associate, Department of Pharmacology and Experimental Neuroscience, College of Medicine, the University of Nebraska Medical Center, USA

Background: Aging is one of the significant risk factors for Alzheimer's disease (AD). However, age-linked cellular events and immune responses that correlate with AD onset and progression remain incompletely understood.

Methods: To unravel relationships between immunity, cell signalling, and aging composite, hippocampal immune transcriptome and regulatory T cell (Treg) numbers and function in blood, spleen, and lymph nodes were assessed by RT2 Profiler Mouse Innate and Adaptive Immune Array and flow cytometric analysis, respectively, in transgenic APP/PS1 mice at 4, 6, 12, and 20 months.

Results: Ingenuity pathway analysis (IPA) comparisons demonstrated significant enrichment in inflammatory, oxidative, and cellular activation pathways. These included triggering receptors expressed on myeloid cells 1 (TREM1), Th1, NF-κB, IL-17, nitric oxide production, acute phase responses, and T cell receptor signalling. Each of these pathways was reduced at 6 months while increased at 12 months. At 20 months, TREM1, pro-inflammatory factors, and NF-κB signalling pathways were increased. We also observed a diminished Treg immunosuppressive function at 6 and 12 months with a restoration of partial immunosuppression at 20 months.

Conclusion: These inflammatory, oxidative stress, and Treg immunosuppressive signatures were linked to progressive AD pathologies and potentially represent targets for immunotherapy in AD.

Biography

Pravin Yeapuri is a post-doctoral research associate in Dr. Howard Gendelman's lab at the University of Nebraska Medical Center, Dept. of Pharmacology and Experimental Neuroscience. His research focusses on evaluating the role of different T cell subsets in neurodegenerative disease and developing T regulatory cell-based therapies for Alzheimer's and Parkinson's disease. Additionally, he is developing humanized, CRISPR based knock-in mouse models for Alzheimer's disease. These models not only replicate the clinical progression of disease but also help evaluate the role of HIV induced neuroinflammation in Alzheimer's disease pathobiology.

Integral Management of the Patient with Epilepsy

Juan E Bender del Busto
Consultant Professor, Project of Epilepsy Surgery at International Center of Neurological Restoration (CIREN), Havana, Cuba

Epilepsy is considered as old as humanity and one of the most frequent disorders of the central nervous system; for some researchers, it is the second neurological disease. It had passed through different cultures and times, known by different names, with a supernatural interpretation of its causes and therefore with the use of various treatments, which included exorcism and trepanation, among others, until the 19th century when Scientific and modern therapy began, which remains in full development to this day, with the incorporation of the therapeutic arsenal and alternative methods described. The affectation in the psychological, social, and community sphere of the patients who suffer from it is significant since they are among the most vulnerable in any society, with the particular stigma that this disease carries with it, which has been transmitted for generations. For this reason, it is intended to describe the aspects that the physician should consider when he is suspected of dealing with a patient with a diagnosis of epilepsy to try to minimize the devastating aspects of this disease. The conference will be based on four main questions: Are we dealing with a patient with epilepsy? What type of seizures/epilepsy/syndrome does he suffer from? What is the cause of the disease? And what behaviour should we follow?

Biography

Juan E Bender del Busto is a Consultant Professor, in the Project of Epilepsy Surgery at the International Center of Neurological Restoration (CIREN), Havana. First Board Member of Cuban League Against Epilepsy (ILAE).

Zein Nerve Conduit for Repair of Large Sciatic Nerve Defect

Guowu Wang[1], Yaofa Lin[2], Yao Xue, Haodong Lin[2] and Jin Ye Wang[1*]
[1]School of Biomedical Engineering, Shanghai Jiao Tong University, China
[2]Department of Orthopaedics, Shanghai General Hospital, Shanghai Jiao Tong University School of Medicine, China
*Shanghai Jiao Tong University Country, China

Peripheral nerve injury is a common clinical issue and challenge to human health that can result from natural disasters, industrial injuries, traffic jams, war wounds, and even some systemic diseases. Grafts such as acellular allogeneic or xenogeneic tissues and artificial conduits must often be implanted when the nerve lesion has a long gap or defect. Among various grafts, autografts are still regarded as the clinical gold standard for repairing nerve injury. However, the limited sources of donor tissue, extra incisions, and the need to sacrifice normal nerve tissue, which leads to the risk of neuroma, limit the use of autografts. Thus, artificial nerve conduits were developed in the 1980s. Initially, these conduits were non-degradable, thereby inducing chronic inflammatory responses and leading to poor functional recovery in the regenerated nerve, often requiring a second surgery to remove these materials. Therefore, improvement using biodegradable materials has been carried out subsequently. Considering the unique properties of zein, such as its biocompatibility, biodegradability, and ease of fabrication, we report the use of zein conduits to repair injured rat sciatic nerves, firstly with a 10-mm defect rat model. Three-dimensional zein conduits were designed with/without pores, and with/without microtubes including in the lumen of conduits. Zein conduit with microtubes yielded satisfactory results in sciatic function index (SFI), proximal compound muscle action potentials, the density of myelinated nerve fibers, and myelin thickness, which were not inferior to autograft but slightly superior to the hollow conduit at the 4th-month post-implantation. The conduits degraded almost completely within two months, which was shorter than the suggested period of four months. Thus, the use of a porous conduit with microtubes inside as guidance may play an important role in successful repair. Next, we challenged the repairment of the 15 mm defect rat model as it is much more difficult to regenerate than the 10 mm defect rat model when no addition of cells and growth factors. Considering the possible body's immune response to this plant protein material, we focused on the design of pore structured conduit and/or encapsulating an anti-inflammatory drug dexamethasone. Zein triggered an early inflammatory response, but this response decreased to the level of the safe materials ALG and PLGA with degradation. Pore structure inhibited neutrophil recruitment and promoted macrophages polarizing towards the M2 phenotype. Thus, porous zein conduits with high and low porosity are further fabricated for the 15 mm sciatic nerve defect repair in rats. The conduits with high porosity induced more M2 macrophages to accelerate nerve regeneration with a shorter degradation period and better nerve repair efficacy.

Biography

Professor Jin-Ye Wang, Ph.D. (Tohoku University, Japan, 1992). Professor at Shanghai Institute of Organic Chemistry, Chinese Academy of Sciences (2000-2009), Professor of Biomedical Engineering, Shanghai Jiao Tong University (2009-). Published 140 papers, 4 books (chapters); authorized 20 patents; invited speaker of European Conference on Biomaterials, Pacifichem et al.; awarded Hundred Talent Program of the Chinese Academy of Sciences (1999), Life Sciences Prize from Meiji Dairies Corporation (2008) et al. Research interests: 3D Bioprinting, Tissue Engineering, Controlled Release, Biomimetic Materials, and Bio interfaces.

NWC 2023

DAY 2

Virtual Presentations

Predictors of Spontaneous Neurological Recovery and Factors affecting Management of patients with Traumatic Spinal Cord Injuries

W S El Masri
Currently Hon. Clinical Professor of Spinal Injuries (SI), Keele University & Emeritus Consultant Surgeon in Spinal Injuries at The Robert Jones & Agnes Hunt Orthopaedic (RJAH)Hospital Oswesty UK

The incidence of Traumatic spinal cord injuries (TSCI) is small and ranges between 10-50/million population/year. Before the second WW, the great majority of patients died within two years of injury. Since the 2nd WW, due to the efforts of the pioneers who dedicated their professional lives to the field of TSCI, most well-managed patients have been able to lead enjoyable, dignified, fulfilling, productive, and often competitive lives, and many depending on the presence of short and long tract sensory sparing exhibit significant degrees of neurological and functional recovery locally or below the level of their injury. To achieve this, however, requires an in-depth understanding of the systemic effects of cord damage on the neurological and functional outcomes and expert simultaneous management of the injury together with the potentially devastating and life-changing medical, physical, psychological, social, financial, vocational, environmental & matrimonial consequences that affect the patient, family members. Knowledge experience and skills in the adequate management of patients with TSCI necessitate training in dedicated Centres that treat all aspects of TSCIs in large numbers and under one roof.

TSCIs cause multi-system physiological impairment and malfunction. This impairment is dynamic and affects the functioning of the various systems of the body during the transitional stage between spinal areflexia and the return of autonomic and spinal reflexes. During this transition, the management of the various systems of the body requires modulation. Following the return of reflex activity, the function of the various systems affected remains at risk of being unstable and erratic. This is because of the various inter-system autonomic and spinal reflex activity caused by the loss of inhibitory and coordinating influence of the higher centers. The combination of an unstable neuro-physiological impairment and sensory impairment/loss can, in inexperienced hands, result in the development of a wide range of potential complications and an increase in disability. Some complications can further damage the Injured and Physiologically Unstable Spinal Cord and cause neurological deterioration, delays, or absence of recovery, imposing further challenges to patients and clinicians. Fortunately, with adequate Active Physio Conservative Management (APCM) of the injury and its medical effects, almost all complications following TSCI can be prevented or diagnosed early and treated before further damage develops.

This necessitates a period of treatment in recumbence until the full return of the autonomic and spinal reflexes. This period ranges between four to eight weeks.

Neurological Recovery can be predicted early in the presence of spared sensory tracts and depending on the extent of the sparing when complications are prevented or diagnosed and treated early. Various groups have repeatedly documented this recovery to occur irrespective of the radiological presentation on X-rays, CT & and MRI since 1969. Unfortunately, it has been rarely referred to in the literature in the last three decades. The last three decades have witnessed increasing claims of benefits of a mechanical interventional approach focusing on the injured spine, often at the expense of the adequacy of management of the medical and non-medical effects of cord injury. Claims that early interventions expedite the mobilization, rehabilitation, and discharge of patients, improve neurological outcomes, or achieve both are currently influencing practice in both well-resourced and under-resourced countries. The risk of further mechanical and non-mechanical damage to neural during or after intervention and during some of the related practices can be potentially detrimental.

I will, in this presentation, discuss the extent of anticipated neurological recovery, the factors that influence its achievement, the role of clinical and radiological findings, and the role of surgery in the short, medium, and long term.

Biography

W S El Masri is Currently Hon. Clinical Professor of Spinal Injuries (SI), at Keele University & Emeritus Consultant Surgeon in Spinal Injuries at The Robert Jones & Agnes Hunt Orthopaedic (RJAH)Hospital Oswestry UK. Has trained between 1971 & 1983 in the Oxford group of hospitals, Guys Hosp. London, Stoke Mandeville Hospital, and the USA. He obtained the first accreditation in the fields of Spinal Injuries and Allied Surgical Specialities in 1982. Appointed Consultant Surgeon in Spinal Injuries at the Midland Centre for Spinal Injuries, the RJAH in 1983. He treated 10,000 patients with Acute Traumatic Spinal Cord Injuries and took full responsibility for the lifelong monitoring and care of 3000 of these patients. He published over 145 manuscripts. He is the author of the: Concepts of "Physiological Instability of the Spinal Cord", "Time related Biomechanical Instability", and "Micro-instability of the injured Spine" and published the largest series of Bladder cancer in SCI patients as well as the longest follow-up manuscript on Post Traumatic Syringomyelia. He has repeatedly demonstrated and published on the discrepancy between the radiological and neurological presentations of patients in support of the hypothesis that the initial force of the impact and the quality of the management of both the injured spine and the effects of cord injury are the two major determinants of the initial neurological loss and the neurological outcome. He is past president of the International Spinal Cord Society; Past Chairman British Association of Spinal Cord Injury Specialists and has lectured worldwide. He is a Founder member of the SPIRIT Educational Charity in Spinal Injuries and is currently Chairman of Trustees of the Charity He won many National and International awards and was commended in the House of Lords on two occasions.

Longitudinally extensive transverse myelitis following SARS-CoV-2 infection

Renate Krassnig*, [1]Gabriela Wittgruber, [2]Christian Enzinger, [1]Werner Lenhard, [3]Gloria Hohenberger
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[2]Medical University Graz, University Hospital of Neurology, Austria
[3]LKH Feldbach-Fürstenfeld, Department of Trauma Surgery, Austria
Consultant of Orthopaedic Surgery and Traumatology, Austria

We report about a 50-year-old previously healthy and very sportive patient who suffered a SARS-CoV-2 Infection in the middle of February 2022 with mild symptoms. About six weeks after the infection, he had problems walking. There was a loss of strength to lift his legs. For this reason, the family doctor sent him for an MRI. The MRI showed a prolapse of the discus at the segment L5/S1 and no myelopathy. The patient consulted the Provincial Hospital, where he was admitted as an inpatient. During the next two weeks, no one found a real reason for the weakness of the patient's legs. After three weeks more, the patient felt worse. Another MRI was performed without any difference from the one before. Empiric therapy with immunoglobulins and corticosteroids was started without any effect. At the end of May, the patient was transferred to the University Hospital Graz, Department of Neurology, with the diagnosis: of Paraplegia Th7 with vegetative disorders. At the Department of Neurology, the therapy with immunoglobulins was started again over a period of 5 days. The next MRI performed on the 3rd of June showed long-distance myelopathy from C2 to Th12 concerning the spinal cord's sides and dorsal strands. The cerebrospinal fluid analysis and antibodies in the blood showed no pathologic findings. According to the Department of Neurology, the etiology of this long-distance myelopathy is still unclear, and there is a high probability of a post-Covid association. On the 21st of August, the patient started rehabilitation at the AUVA Rehabilitation Clinic Tobelbad. On the 10th of October, the patient said, for the first time, that he could move his toes a little bit and he was feeling pressure in his bladder and rectum. If he develops more functions, we will follow up.

Biography

Renate Krassnig was born on the 15th of January in Carinthia/Austria. She finished University in 2006 and started her Residency at the University Hospital of Trauma Surgery. Till 2019 she worked at the Medical University Graz and published numerous articles and gave more than 100 talks. Since 2019 she has been a Consultant at the AUVA Rehabilitation Clinic in Tobelbad. She is a Consultant of Orthopaedic Surgery and Traumatology and takes care of the paraplegics.

The Tragic Migraine Classification Fiasco

Elliot Shevel
Medical Director, The Headache Clinic, South Africa

Objective: To expose fatal flaws in the International Headache Society's (IHS) International Classification of Headache Disorders (ICHD) in the diagnosis of migraine.

Objective: To expose fatal flaws in the International Headache Society's (IHS) International Classification of Headache Disorders (ICHD) in the diagnosis of migraine.

Background: The ICHD is universally accepted by researchers and clinicians. It is highly unlikely that specialist headache journals will accept submissions for publication if the cohorts have not been selected strictly according to the ICHD diagnostic criteria. Likewise, in the clinical setting, the appropriate treatment is prescribed according to how the patient's headache is classified in the ICHD. If, however, the ICHD is not substantiated by scientific data, then it is not possible for research based on the ICHD to be regarded as "evidence-based".

Methods: A detailed analysis of the bibliography and references in the "Migraine without Aura" section of the ICHD, and a determination of whether the ICHD is based on sound scientific data.

Results: There are 13 references in the "Migraine without Aura" section of the ICHD-3, the latest version of the ICHD, published in 2013[1]. None of these contain data substantiating the choice of criteria for the diagnosis of migraine. The same applies to the ICHD-1 (1988) and the ICHD-2 (2004).

Conclusion: Although the references analyzed are only from the migraine without aura section of the ICHD, the same principles apply to all the other sections of the ICHD relating to primary headaches. Professor Jes Olesen, who was chairman of the CC from its inception in 1985 till the ICHD-3 was published in 2018, admitted that the IHS diagnostic criteria are "based on opinions" and are "not based on empiric data".[2] The only possible conclusion is that migraine research and treatment since 1988, based on the ICHD, has no scientific validity.

Biography

Dr. Shevel has gained international recognition for his development of highly effective techniques for the treatment of migraines, tension headaches, and cluster headaches. Presented papers at International Conferences in 12 countries with particular reference to migraine and has had more than 40 papers published in peer-reviewed medical journals.

The True Trigeminovascular System

Elliot Shevel
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The trigeminovascular system, comprising the trigeminal subnucleus caudalis, the trigeminal nerve, and the dural meningeal arteries, is widely accepted as playing a fundamental role in the pathogenesis of migraine. That the dural arteries are involved in migraine has however never been proven and is based solely on animal studies and speculation. In this article, evidence is presented to show that the dura mater and its vasculature are not in any way involved in the pathogenesis of migraine and that the true vascular element of the trigeminovascular system is the extracranial terminal branches of the external carotid artery.

Biography

Dr. Shevel has gained international recognition for his development of highly effective techniques for the treatment of migraines, tension headaches, and cluster headaches. Presented papers at International Conferences in 12 countries with particular reference to migraine and has had more than 40 papers published in peer-reviewed medical journals.

Neurophysiological Studies to Counteract the Oxidative Stress in Age-Related Macular Degeneration

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Associate Professor of Physiology at the University of L'Aquila

As part of the central nervous system, the retina is particularly susceptible to alterations in its microenvironment, which can cause irreversible damage to vision. In addition, the retina, and especially the macula, is characterized by a state of physiological oxidative stress due to an elevated metabolism and high oxygen consumption. Hence, the maintenance of a correct and balanced microenvironment is fundamental to the health of the retinal cells. Age-related macular degeneration (AMD) occurs because of photoreceptors, retinal pigmented epithelium, Bruch's membrane, and choriocapillaris complex alterations, which culminates in blood-retinal barrier breakdown, activation of inflammatory events, and retinal neurodegeneration. AMD can be considered a multifactorial disease and the main risk factors include aging, cigarette smoke, high-fat diet, light exposure, alcohol consumption, and specific genetic polymorphisms. All these events share oxidative stress as a common feature that can be considered the driving force of all the risk factors. Based on the absence of effective therapies for the treatment of AMD, in recent years important experimental approaches have focused on nanomedicine, which represents a promising research field due to the unmatched properties of nanoparticles. Cerium oxide nanoparticles (CeO2-NPs), a pure antioxidant, have been tested in our animal model of AMD and we demonstrated that the main features of AMD can be counteracted. Specifically, we have demonstrated their ability to preserve retinal function and avoid the blood-retinal barrier breakdown and debris accumulation, to counteract neovascularization and microglial activation. Based on this evidence, it can be taken into consideration that CeO2-NPs may represent a promising therapeutical approach for AMD.

Biography

Rita Maccarone's research field is focused on the functional study of the visual system and the mechanisms underlying retinal neurodegeneration. In particular, the scientific interest is focused on the degeneration of the neuroretina and retinal pigment epithelium, the involvement of the retinal vascular system, and the breakdown of the haematoretinic barrier. The experimental approach consists of in vitro and in vivo studies to broaden the knowledge of the processes that lead to vision loss in human diseases such as age-related macular degeneration with the aim of.

For many years she shared her interest in the physiopathology of the visual system with international research groups. She had established collaboration with Prof. J. Stone and Prof. Kristztina Valter, of the "Australian National University", Canberra, and the University of Sydney and with Prof. Kristrof.

She participated in several funded research projects (NATO Italy-Poland 2001, ASI 2001, CNR 2001, Boehringer 2002, PRIN 2000-2004-2006, POR Abruzzo 2004, International collaboration Italy - France 2005, Telethon)

Comprehensive Characterization of Perinatal Oxycodone-exposed Offspring in Early Adolescence

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The study of the opioid epidemic is often limited to acute health problems and neglects perinatally impacted offspring. Treating these offspring represents a new impending public health challenge that current healthcare systems do not know how to address. Literature suggests significant neurodevelopmental and behavioural deficits between offspring exposed to oxycodone (OXY) in utero (IUO) and post-natal (PNO) in early life and later adulthood. However, early adolescence represents a significant gap in our understanding of PNO- and IUO-offspring. We hypothesized that PNO- and IUO-offspring are primed to experience developmental deficits during adolescence. To test this hypothesis, our study employed an integrated systems approach evaluating phenotype, molecular, and behavioural impacts on PNO- and IUO-offspring. Phenotypic measurements of PNO- and IUO-offspring show OXY-induced significant reductions in head size, brain weight, and body composition. At the same time, our molecular studies found a lower transcription of inflammasome-specific genes in the prefrontal cortex of exposed offspring. Phenotypic and neurological deficits paired with our behavioural studies show that PNO- and IUO-offspring are primed for heightened anxiety-like behaviour under social stress. In summary, our study, for the first time, has performed a comprehensive analysis of how perinatal OXY exposure impacts outcomes in both the PNO and IUO-offspring during early adolescence.

Biography

Dr. Pendyala is a Robert Lieberman MD Ph.D. Endowed Professor in the Department of Anaesthesiology at the University of Nebraska Medical Center (UNMC). His research program focuses on Addiction, Neurodevelopment, Infectious diseases, and Environmental toxins. In addition, over the last three years, he has been actively engaged in elucidating the role of biopsychosocial risk factors in pregnant women and developmental outcomes in newborns. Dr. Pendyala is a recipient of several awards that include being elected as a life member of the Indian Academy of Neurosciences, being Recipient of the Joseph P. and Harriet K. Gilmore Distinguished New Investigator Award for outstanding research awarded by the Dean's office, Innovators of the Year Special Award and recently inducted into the "Circle of Distinction" conferred from the Chancellor of UNMC.

Towards A General Brain Activity Theory: The Biological (Social) Expediency of the Physical Activity of Neuronal Networks is Provided by the Informational and Causal Properties of Mental Phenomena

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In our opinion, modern brain science has stalled on a problem that it has not yet had time to formulate clearly. I formulate this problem in the following question, which reveals a paradigmatic contradiction in our knowledge about the informational activity of the brain. Indeed, on the one hand, our brain, with all its neuro-electrical, neuro-molecular, neuro-ionic, and structural-network properties, is, by and large, a physically existing and physically functioning entity. However, on the other hand, its informational activity, for some reason, "paradoxically" happens biologically (and later in evolution, socially) expediently, as if the brain's informational processes obey some unknown management factor that takes care of the biological (or social) problems of the existence of living beings. So, the question of what makes the "physics" of the brain process information in such a way (and we know that not one physical law support any biologically expedient process) is a key question, the answer to which could completely change the paradigm of our understanding of how the brain works [1]. The short (but not complete) answer to this question is as follows: neural networks of the brain process information biologically (or socially) appropriately because mental phenomena are formed in its limbic region, which realize the ability of living beings to subjectively evaluate biological (or socially) expediency of information about the surrounding world. It is this ability to subjectively evaluate the expediency of external stimuli and information about them during the elimination of uncertainty arising in the relationship of a living being with the environment that is the key aspect that creates mental motivation for self-management by a living being of all informational operations in the brain [2]However, this short answer to the question formulated here, to explain the way of the causal inclusion of mental phenomena in the physical activity of the brain, should be supplemented by the following statement. It consists of the following: the ability of our brain to evaluate information through subjective evaluation forms hierarchical relationships in the brain between functionally specific brain structures and creates conditions for the integration of information (G. Tononi) within such Bottom-Up & Top-Down activity [1]. During such activity, management functions are performed precisely by the structures of the limbic region, which realize the ability of a living being to subjectively evaluate something to integrate (combining) fragments of experience into more complex information concepts that fix regularities in their structure. Through these more complex mental informational concepts (mental images, thoughts), control neural networks orchestrate physical processes in subordinate brain structures that directly implement the processes of fixation, integration, and embodiment of information in "informational–well-enriched" motor acts. So, in this way, a living being with an appropriately developed brain under conditions of "pressure" of uncertainty transforms around the world through arbitrary movements from a state of "subjectively undesirable" to a state of "subjectively desirable" [1]. Of course, for a neurophysiologist who is used to studying the brain within the paradigm of classical physical determinism, such a solution to the problem, which is considered here within the framework of a paradigm where mental phenomena formed in control neural networks can orchestrate physical processes in networks controlled by them, is very unusual. But if we need a breakthrough, then let be the breakthrough.

Biography

Oleg Solovyov is a Full Professor in the Department of Psychology, Pedagogy, and Philosophy of Kremenchuk National University (Ukraine). Before that, he headed the Department of Human Health at the East Ukrainian National University. In 2012, he received his PhD (Psychophysiology). For 17 years, he worked in the children's city hospital in Luhansk, specializing in rehabilitating cerebral palsy and other diseases associated with neurological disorders of motor activity.

Depression Diagnosis Based on Electroencephalography Power Ratios

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Depression is a common mental disorder that impacts millions of people worldwide. However, its diagnosis is difficult owing to subjective testing. Although quantitative electroencephalography (EEG) has been investigated as a promising diagnostic tool for depression, the associated results have proven contradictory. The current study seeks to determine whether the alpha/beta, alpha/theta, and theta/beta ratios can be biological markers of depression. We used open-access EEG data to investigate these power ratios in 46 patients with depression and 75 healthy individuals. Decreased anterior frontal, frontal, central, parietal, occipital, and temporal alpha/beta ratios and decreased central and parietal theta/beta ratios were observed in the depression group. All ratios could effectively differentiate depression from healthy individuals. In particular, the central, frontal, and parietal alpha/beta ratios exhibited high discriminating ability. No significant differences were observed in the alpha/theta ratio. Depression, stress level, and the probability of depression increased with a decrease in the central alpha/beta ratio. Moreover, machine learning analysis revealed that the alpha/beta ratio was the most effective for diagnosing depression among the three EEG power ratios. These findings lay the foundation for future studies to develop more effective and efficient methods for diagnosing depression.

Biography

Jinwon Chang is currently a student-researcher at the Korean Minjok Leadership Academy. I love studying neuroscience and conducting experiments with open-access data. Despite lacking a specific degree, I investigate how brain waves can be used to diagnose dementia or other cognitive disorders. Using high-quality open-access data, I'm good at drawing new conclusions with different perspectives from previous neuroscience projects. I frequently examine various scientific studies and methods in a school laboratory by cooperating with colleagues.

Genomic Characterization and Molecular Mechanisms in Glioblastoma Multiforme and Current Treatment Strategy

Saeede Zarei
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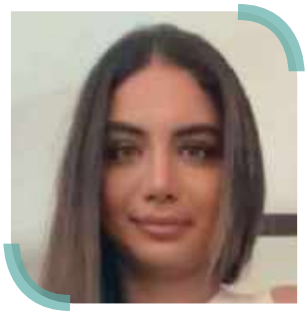

Glioblastoma (GBM) is the most aggressive malignant primary brain cancer, with an incidence rate of 3.9 per 100,000 persons in the United States and a median age of 64 years. GBM has an average life expectancy of one year. This is mostly due to the highly muted genome of GBM, which is characterized by the deregulation of many key signalling pathways involving growth, proliferation, survival, and apoptosis. In this review, I try to summarize the molecular mechanisms that have led to the GBM and discuss the potential therapeutic strategies and approaches.

Biography

Saeede Zarei graduated with a master of pharmaceutical science (pharmacology and toxicology) from Pharmacy College of Long Island University in Brooklyn, NYC. Then, I moved to LOS Angeles and started my career as QC lab lead at a pharmaceutical company. My interest and research work are in brain cancer.

Coma and Disorders of Consciousness in Stroke

Shraddha Mainali
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There has been a tremendous improvement in the outcomes of stroke over the past 2 decades. Novel technologies and management strategies have improved the lives of millions of people with stroke globally. Apart from improved treatment strategies, the novel diagnostic, and prognostic tools such as the ability to discern cognitive motor dissociation in apparently comatose patients have provided hope for clinicians and families of patients with acute brain injury. The launch of the Curing Coma Campaign by the Neurocritical Care Society is a major step forward in improving the outcomes of comatose patients. It is time to awaken hope and collaborate globally to improve the lives of patients with coma and disorders of consciousness.

I will speak on the history of coma and Doc, outcomes associated with stroke, and the way forward with focus on initiatives of the Curing Coma Campaign which is a global network of over 300 clinicians and coma scientists across the world.

Biography

Dr. Mainali is a neurointensivist and a stroke neurologist and an Associate Professor of Neurology at Virginia Commonwealth University (VCU). She is the Director of Clinical Research for Stroke and Neurocritical Care at VCU. She serves on the executive committee of the Curing Coma Campaign as well as on the scientific advisory committee and the scientific steering committee. She is the Chair of the Research Operations Subcommittee of the Neurocritical Care Society. She has been an invited speaker on national and international topics related to stroke, coma, disorders of consciousness, and Transcranial Doppler Monitoring in the ICU. She serves on the editorial board of the Neurocritical Care journal and serves as a guest editor for other journals such as Seminars in Neurology and Frontiers.

Comparison Between Supraorbital Keyhole and Kocher's Point Approach as an Optimal Trajectory for Stereotactic Hematoma Aspiration of Basal Ganglia Haemorrhage: Clinical Analysis and Computational Simulations

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Introduction: Regarding the recent trials, minimally invasive techniques of frameless stereotactic hematoma aspiration is known as a safe and effective method to remove acute intracerebral haemorrhages (ICH), with the potential to enhance neurological recovery. Supraorbital keyhole (SOK) surgeries for the anterior skull base approach were widely used, and the ventriculostomy via the basal frontal lobe was attempted with acceptable safety and feasibility. Considering the anatomic location and shape of basal ganglia, catheter placement via SOK can have a larger length within the hematoma and can lead to better aspiration rate and functional outcomes compared to the conventional Kocher's point (KP) approach; however, no studies on this topic exist. Verifications with retrospective clinical data and computational simulation were attempted.

Methods: We retrospectively enrolled 76 patients who underwent hematoma aspiration of basal ganglia ICH and divided them into KP and SOK groups. Clinical comparison between the groups and subgroup analysis within typical basal ganglia ICH patients (n=50) were performed. In 8 cases, computational simulation of hematoma aspiration was conducted with preoperative 2mm thinly sliced brain computed tomography images.

Results: In comparison analyses between KP and SOK, the aspiration rate of the hematoma was much lower in SOK groups (50.71% vs. 38.49%); however, the significance was not observed (p=0.077). In subgroup analysis, the aspiration rate was significantly higher in the SOK group than that of the KP group (52.6% vs. 33.6%, p=0.0391), and good functional outcome (modified Rankin Scale score: 0 to 2) was significantly higher in the SOK group (71.4% vs. 28.6%, p=0.049). In computational simulations, residual volume was much lower in SOK patients with a typical type of ICH.

Conclusion: Stereotactic hematoma aspiration via the SOK route in patients with the typical shape of basal ganglia ICH was identified as a safe and effective method with enhancing aspiration rate and favourable functional outcomes. In computational simulations, residual volume was much lower in typical basal ganglia ICH patients.

Biography

Jang Hun Kim is working as a Clinical Associate Professor at the Department of Neurosurgery at Korea University Anam Hospital, Korea University College of Medicine. He completed his Master's (Neurosurgery) at the Graduate School of Medicine, Korea University, Seoul, Korea. He worked as a Clinical Professor, Neurosurgery, at Seoul National University Bundang Hospital

The Effect of Two Modalities of Sleep Disruption on the Immune System in Healthy Female Participants

Zuha Ajlan
Master of Science in Medicine candidate at the University of the Witwatersrand, South Africa

Sleep and immunity have a bidirectional relationship needed for homeostatic regulation. Studies have shown that sleep deprivation dysregulates the immune response by elevating pro-inflammatory markers, interleukin (IL-)1, IL-6, and tumor necrosis factor (TNF-)α and increases the susceptibility to autoimmune disorders. Despite women being 80% of all autoimmune patients and having a greater prevalence of sleep disorders, most experimental human studies focused on men. Therefore, the study assessed the effect of acute sleep disruption on sleep parameters and immune response in healthy young women.

Fourteen healthy females underwent three randomized, sleep conditions: baseline night (BN) – uninterrupted 8 hours of sleep; restriction night (RN) – sleep was limited to the first 4 hours and awake for the remaining 4 hours; fragmentation night (FN) – eight randomized forced awakenings through an 8-hour sleep opportunity night. Polysomnography was conducted for each condition and plasma was collected. A multiplex Luminex was measured cytokines: IL-1β, IL-2, IL-4, IL-6, IL-8, IL-10, IL-17a, TNF-α and IFN-ɣ.

The study found a significant effect of sleep conditions on IL-2 concentration. There was a significant effect of TST, WASO, and global NREM on daytime IL-8 concentration following sleep conditions. While sleep conditions did not significantly affect IL-8, there was a trend of an increase in IL-8 between conditions ($F = 3.40$, $P = 0.051$). Moreover, N2 significantly decreased ($F = 6.28$, $\beta = -0.004$, $P = 0.02$) and N3 ($F = 7.01$, $\beta = 0.006$, $P = 0.0151$) increased TNF-α following sleep deprivation.

The study shows that acute sleep disruption disrupts the immune system and alters sleep architecture. Further research is needed to investigate the underlying mechanism and potential treatments for autoimmune disorders in women.

Biography

Zuha Ajlan is a Master of Science in Medicine candidate at the University of the Witwatersrand focused on neurophysiology, immunology, sleep medicine, and women's health. She is a compassionate, resourceful, and resilient young researcher with a passion for neuroinflammation and women's health.

Towards Quantitative Perfusion MRI of the Lung in COPD: The Problem of Short-Term Repeatability

Alvard Ter-Karapetyan
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Purpose: 4D perfusion magnetic resonance imaging (MRI) with intravenous injection of contrast agent allows for a radiation-free assessment of regional lung function. It is, therefore, a valuable method to monitor response to treatment in patients with chronic obstructive pulmonary disease (COPD). This study was designed to evaluate its potential for monitoring short-term responses to hyperoxia in COPD patients.

Materials and methods: Nineteen prospectively enrolled COPD patients (median age 66y) underwent paired dynamic contrast-enhanced 4D perfusion MRI within 35min, first breathing 100% oxygen (injection 1, O2) and then room air (injection 2, RA), which was repeated on two consecutive days (day 1 and 2). Post-processing software was employed to calculate mean transit time (MTT), pulmonary blood volume (PBV), and pulmonary blood flow (PBF), based on the indicator dilution theory, for the automatically segmented whole lung and 12 regions of equal volume.

Results: Comparing O2 with RA conditions, PBF and PBV were found to be significantly lower at O2, consistently on both days ($p<10–8$). Comparing day 2 to day 1, MTT was shorter by 0.59±0.63 s ($p<10–8$), PBF was higher by 22±80 ml/min/100ml ($p<3·10–4$), and PBV tended to be lower by 0.2±7.2 ml/100ml ($p = 0.159$) at both, RA and O2, conditions.

Conclusion: The second injection (RA) yielded higher PBF and PBV, which contradicts the established hypothesis that hyperoxia increases lung perfusion. Quantification of 4D perfusion MRI by current software approaches may thus be limited by residual circulating contrast agents in the short term and even the next day.

Biography

I'm a radiologist at Vardanants Center of Innovative Medicine in Yerevan. I have studied at Yerevan State Medical University. After graduation, I researched at the University Hospital of Heidelberg, Germany. In 2019 I participated in ECR in Vien, where I presented my research. I have also participated in many conferences. The last one was in Orlando one month ago.

Lateralization Manifestation of the Left Medial Limbic System in Mild Cognitive Impairment with Neuropsychiatric Symptoms

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Alzheimer's disease (AD) is a neurodegenerative disease with a non-linear pathophysiological dynamic, culminating in symptomatic late stages with cognitive and behavioural decline. Amyloid-β (Aβ) and tau proteinopathies are the neuropathological hallmarks of AD.

Neuropsychiatric symptoms (NPS) such as depression, apathy, aggression, psychosis, and sleep-wake disturbances are core features of AD alongside prominent memory loss. Neuropsychiatric symptoms (NPS) have been increasingly detected as early non-cognitive indicators of the AD continuum. We investigated AD pathology at the prodromal symptomatic stage, under NPS, before converting to AD dementia.

A total of 139 elderly individuals were stratified based on the global depression scale (GDSCALE), neuropsychiatric inventory total scores (NPITOT), clinical dementia rating (CDR), and mini-mental status examination (MMSE), into 12 mild cognitive impairment (MCI) individuals with NPS (MCI+) and 49 without NPS (MCI-), 6 AD dementia patients with NPS (AD+), 23 AD individuals with NPS (AD-) but no cognitive impairment, and 49 age and sex-matched normal individuals.

MRI-based Voxel-Based Morphometry (VBM) and DTI-based Tract-Based Spatial Statistics (TBSS) were first used to analyze brain-wide group differences. Second, the graph theoretical approach was used to investigate brain structural connectivity alterations. Moreover, correlational tractography was performed to identify White Matter (WM) diffusion metrics associated with AD CSF and plasma biomarkers.

VBM showed significant volume reduction only in MCI+ individuals compared to normal controls in the left Hippocampus, Amygdala, and primary sensory cortex (SSC). No WM or Gray Matter (GM) abnormalities were detected in the MCI- group.

But in contrast to the VBM, structural connectivity estimation revealed structural WM connectivity loss in both hemispheres of MCI- individuals relative to normals, including the Thalamus, Pallidum, Putamen, Caudate, and hippocampus on the left side and SSC and frontal cortex on the right side, indicating limbic system involvement before overt volumetric GM loss.

Biography

Shahryar Pajavand is working as a Researcher at Shahid Beheshti University of Medical Sciences Skull Base Research Center (SBRC), Iran. Shahryar Pajavand is a dedicated and passionate researcher in the field of neuroscience. As a Ph.D. candidate in Neuroscience, Shahryar has developed a strong foundation in neuroimaging, precision medicine, and glioma tumors. With expertise in conducting research studies and analysing data using advanced techniques such as diffusion tensor imaging, fMRI, Python, and MATLAB, Shahryar is committed to exploring the intricacies of Alzheimer's disease and its impact on the brain. With a keen interest in unravelling the complications of the human brain, Shahryar is excited to present his work and share his findings at the upcoming conference, contributing to the advancement of knowledge in the field of neuroscience.

The Effect of Group Psychomotor Therapy on Self-perception of Ability and Social Acceptance of Children 5-6 Years Old

Georgios Moschos
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Group Psychomotor Therapy (GPT) has a positive effect on the development of preschool-age children. GPT is prescribed for children with behavioural problems and has been used to enhance motor competence. The purpose of the study was to examine the effect of a GPT on the self-perception of ability and social acceptance of children 5-6 years of age. In the survey participated, 81 children aged 47-72 months attended private Kindergartens in Ioannina, Greece. The sample was assigned to the Experimental Group (EG), which participated in the GPT program, and the Control Group (CG), which did not participate in any intervention program. Children were measured with the Pictorial Scale of Perceived Competence and Social Acceptance (PSPCSA) in the Greek version. According to the results, children of the EG presented higher improvement in self-perception of ability and social acceptance compared to those of the CG. GPT plays an important role in the child's development and is recommended in schools and kindergartens. The results confirm that GPT affects not only the emotional profile but also the reduction of behaviour problems. It is important to include GPT in the prescription procedure within the Greek Health System to provide additional support in traditional methods and treatments.

Biography

Georgios Moschos is a PhD-student. His research topic is the effect of a Psychomotor Intervention program on motor, social, emotional, and school competence of children aged 4-5 years old without any diagnosed neurological, sensory, or motor impairments. His scientific specializations are motor development, psychomotor intervention, and intellectual-developmental disabilities. He is also a teacher of psychomotor intervention at the kindergarten in Ioannina, Greece.

Advances in Focused Ultrasound for the Treatment of Brain Tumors

Brandon Lucke Wold
Co-Founder, Wright-Wold Scientific, USA

Advances in Focused Ultrasound for the Treatment of Brain Tumors

Brandon Lucke Wold
Co-Founder, Wright-Wold Scientific, USA

The arsenal of therapeutics for brain tumors is limited by the relative impermeability of the blood-brain and blood-tumor barriers. In physiologic states, the blood-brain barrier (BBB) serves a protective role by passively and actively extruding neurotoxic compounds. However, this function limits the penetrance of therapies into the tumor microenvironment (TME). Advances in focused ultrasound (FUS) technology provide a method of bypassing the BBB through ultrasound frequency to transiently permeabilize the BBB and BTB. Concomitant delivery of tumor therapeutics has allowed for previously impermeable therapeutics to reach the TME. This review details the advances in FUS in both preclinical and clinical models, focusing on its safety profile. We then turn the discussion toward future directions in FUS-mediated therapies for brain tumors. Keywords: Microbubble-enhanced focused ultrasound, blood-brain barrier, blood tumor.

Biography

Brandon Lucke-Wold was born and raised in Colorado Springs, CO. He graduated magna cum laude with a BS in Neuroscience and distinction in honours from Baylor University. He completed his MD/Ph.D., Master's in Clinical and Translational Research, and the Global Health Track at West Virginia University School of Medicine. His research focus was on traumatic brain injury, neurosurgical simulation, and stroke. At West Virginia University, he also served as a health coach for the Diabetes Prevention and Management program in Morgantown and Charleston, WV, which significantly improved health outcomes for participants. In addition to his research and public health projects, he is a co-founder of the biotechnology company Wright-Wold Scientific, and the pharmaceutical company CTE Cure, and was a science advocate on Capitol Hill through the Washington Fellow program.

He has also served as president of the WVU chapters for the American Association of Pharmaceutical Scientists, the Neurosurgery Interest Group, and the Erlenmeyer Initiative Entrepreneur group. In addition, he has served as vice president for the graduate student neuroscience interest group, Nu Rho Psi Honor Society, and medical students for global health. He was an active member of the Gold Humanism Honor Society and Alpha Omega Alpha Honor Society. He is currently a member of the UF House Staff Council, Positive Culture Committee, Quality Improvement Committee, Board of Directors Alachua County Medical Society, and Accreditation Requirements Review Committee. He is married to Noelle Lucke-Wold and has two children. As a family, they enjoy running with their dogs, rock climbing, and traveling. In his spare time, Brandon frequently runs half marathons and 10ks together with his wife. Brandon also enjoys reading, playing piano, discussing philosophy, and playing chess. He is currently a Pgy5 neurosurgery resident at the University of Florida with pursuing endovascular enfolded training and was awarded the Dempsey Cerebrovascular Research Fellowship.

Transient Muteness Followed by Dysarthria with Pontine Ischemic Stroke

Dahab Ouhabi
University Hospital Ibn Sina, Morocco

Introduction: Diaschisis is a mechanism describing a decreased function in nervous system regions that are remote from the area of initial damage, but that are connected to the primary site of damage through neuroanatomical pathways.

Methods: Our study is a case report of a patient who was admitted to the emergency room after the sudden onset of pure motor hemiplegia with a transient muteness that only three days later evolved into dysarthria, secondary to a pontine stroke.

Results: A 53-year-old right-handed woman with a past medical history of blood hypertension was admitted to our department ten hours after the sudden onset of pure motor hemiplegia with muteness. On neurological examination, the patient was mute, yet alert and cooperative. There was a right flask hemiplegia with a right central facial palsy, with no sensory disturbance or coordination of the left limbs.

Brain Magnetic Resonance Imaging showed a left paramedian and lateral pons acute ischemic stroke on diffusion and FLAIR weighted images.

On the third day, the patient's mutism evolved into a labored slurring in line with dysarthria.

Conclusion: The Brainstem gathers a rich network of tracts. Cases of transient mutism followed by dysarthria have already been described after surgery of the posterior cranial fossa, but few studies focused on sub-tentorial diaschisis, especially those following a stroke.

Biography

Dahab Ouhabi is a Resident Department of Neurology B and Neurogenetics, University Hospital Ibn Sina, Rabat, Mohammed V University in Rabat, Morocco

Epidemiologic and Clinical Characteristics of Neuromyelitis Optica Spectrum Disorder Patients, A Seven Year Follow-up Study

Roshanak Mehdipour
Isfahan University of Medical Science, Iran

Background: Neuromyelitis Optica Spectrum Disorder (NMOSD) is a rare neuroinflammatory disease characterized by recurrent relapses. The most common signs are myelitis and optic neuritis. Cerebral or brain stem syndromes can also present them. There are still many challenges in its diagnosis and treatment. Indeed, it needed long-term follow-up studies to see the disease course over time.

Methods: We established an electronic registration system for NMOSD patients in October 2015 at Kashani Hospital, Isfahan, Iran. Every suspected patient was documented and included in the follow-up system to survey their disease course. Anti-aquaporin 4 (AQP4) antibody checked for all by cell-based assay method. All information, such as demographic data, history, addiction laboratory, MRI findings, and disease presentation, had been documented. We followed them regarding relapse, new paraclinical tests, and drug changes. This study is based on definite NMOSD cases (according to the 2015 criteria) characteristics and clinical course during seven years of registration.

Results: The study included 173 NMOSD cases, and 56 were seropositive for AQP4 Ab. Their mean age was 40.02±11.11 years (45.78 in the seropositive group). The mean age at disease onset was about 30.16 years. The mean time of follow-up by our registration system is 55.84 ±18.94 months (54.82 months in seropositive ones). The annual relapse rate is estimated as 0.47±0.36. Long-extended transversal myelitis (LETM) was presented in the baseline MRI of 77 patients (44.5%), while 32 of them didn't show any related clinical symptoms. One hundred twenty-four patients revealed an abnormality in the first brain MRI. 27 individuals suffer hypothyroidism as the most common comorbid disease. The disease seems to be more prevalent in Isfahan province's west and southwest areas.

Conclusion: The mean age of onset is higher than Multiple Sclerosis (MS) patients, but there were noticeable paediatric cases too. It should be considered that LETM can be asymptomatic at first. Brain MRI abnormalities are the most prevalent. The disease is more prevalent in geographical areas like MS prevalence.

Keywords: Neuromyelitis Optica, Rituximab, Relapse Rate, Multiple Sclerosis, Cervical MRI

Biography

I am Roshanak Mehdipour, MD, Neurologist from Iran. I was born In March 1987. I studied medicine at Isfahan University of Medical Sciences and graduated as a neurologist in 2017. I was a post-doctoral research fellow in neuroimmunology at Isfahan Neuroscience Research Center between 2017 and 2020. My specific research fields are neuromyelitis optica (NMOSD) and Multiple sclerosis (MS). I have been the director and manager of the NMOSD patient's registry system in our referral MS center since 2015. I have published several articles in this area. I had many presentations (oral / posters) at international scientific congresses. Now I am practicing as a consultant neurologist in a general hospital along with my research activities.

Visual Evoked Potential in Attention Deficit Hyperactivity Disorder Patients Treated and Untreated with Psychostimulant Drugs

Zeynep Unluturk
Clinical Neurologist Turkey

Objective: Attention deficit hyperactivity disorder (ADHD) is a persistent, genetically inherited neurobehavioral and neurodevelopmental disorder that is explained by a variety of neurobiological pathways. The purpose of this study is to compare visually evoked potentials (VEP) latency and amplitude in ADHD patients. It is aimed to discuss the effects of disease severity and psychostimulant use on the central nervous system based on VEP measurements.

Methods: The study included 30 patients, 15 of whom are on psychostimulant drugs and 15 of whom are not. Adult ADHD Diagnostic Screening and Rating Scale were applied to all participants, and VEP measurements were recorded. P100 amplitude and latans were recorded for both eyes. In ADHD patients, these values were compared based on the severity of the disease and of those who receive treatment and those who do not by using Statistical Package for Social Science (IBM SPSS Statistics 25 software).

Results: There was no statistically significant difference between latency and amplitude values according to disease severity (p>0,05). The difference was statistically significant between P-100 latency according to psychostimulant use (p=0,046, p=0,016). Regarding the use of psychostimulants, there was no significant difference in the amplitude values (p>0,05).

Conclusions: Increased sample size VEP studies in ADHD with additional confounding variables will provide insight into the information processing process and shed light on the pathophysiology of ADHD

Biography

Zeynep Ünlütürk was born in Turkey in 1989. She graduated from medical school in 2013 and completed her neurology residency in 2020. She is currently working as a clinical neurologist. Her specialties are neurophysiology, neuromuscular diseases, and neuropathic pain.

A Unique Case of Acute Motor Sensory Axonal Neuropathy with Possible Overlap with Wernicke's Encephalopathy

Sondos Eladawi
Walsall Manor Hospital, UK

Acute Motor Sensory Axonal Neuropathy (AMSAN) is a rare subtype of Guillain-Barré syndrome (GBS), which is an autoimmune disorder that affects the peripheral nervous system. In AMSAN, the immune system attacks the axons of the peripheral nerves, resulting in damage to both the motor and sensory nerves. This leads to muscle weakness, loss of reflexes, and sensory disturbances such as numbness, tingling, and pain in the limbs. Diagnosis of AMSAN is based on a combination of clinical symptoms and nerve conduction studies which help to distinguish AMSAN from other subtypes of GBS, such as acute inflammatory demyelinating polyradiculoneuropathy (AIDP).

The case we present is a unique case of a twenty-four-year-old female who presented to the emergency department with abdominal pain and vomiting. She had a past medical history of Anxiety, depression, polycystic ovary syndrome, and cannabis abuse. She was a current smoker but denied excessive alcohol intake. Six days into admission she was found to have pins and needs in her lower limbs which then progressed to total loss of motor and sensory function in the upper and lower limbs within two weeks. A few days later the patient developed bilateral ophthalmoparesis, nystagmus, and increased confusion. Differential diagnosis considered were Wernicke's encephalopathy, Miller Fisher syndrome, Neuromyelitis optica spectrum disorder, and Central pontine myelinolysis. Laboratory tests were unremarkable apart from mildly deranged liver function tests. She had Magnetic Resonant Imaging (MRI) of the brain and whole spine done which were unremarkable as well. However, nerve conduction study findings were in keeping with AMSAN. She was started on intravenous immunoglobulins (IVIG) and pabrinex to cover for possible overlapping Wernicke's encephalopathy. She then started to improve slowly following the completion of the treatment course and was discharged to a rehabilitation center for recovery. The key to improvement of the long-term outcomes after the acute phase of AMSAN is early initiation of treatment which necessitates early recognition of the disease pattern.

Biography

Dr. Sondos Eladawi MBBCh, MRCP (UK), is a third-year internal medical trainee within the West Midlands Deanery, United Kingdom. She is currently working in the General medical department at Walsall Manor Hospital. Dr. Eladawi has a strong passion for Neurology. She has a special interest in disorders of peripheral nerves, Motor Neuron Disease, and Epilepsy.

Juvenile Myasthenia Gravis: A Case Report

Ishmeet Kaur Jaggi
University College of Medical Sciences and GTB Hospital, India

An eight-year-old female child presented to the paediatric emergency with complaints of generalized muscle weakness and ptosis with diplopia. A patient complained of painless fatiguability of bulbar and limb musculature resulting in dysphonia and difficulty in swallowing. The patient also had proximal limb weakness manifesting as difficulty in getting up from the floor, running, going upstairs, and lifting her arms above her head. The weakness was fluctuant, which became more pronounced on activity and improved on rest. The patient was being worked up for the cause of such weakness when suddenly the patient went into myasthenic crisis in the form of worsening dyspnoea, drowsiness, fatigue, worsening bulbar dysfunction, and marked global weakness with impending respiratory failure. The patient was kept on NIV support and was given intramuscular neostigmine after atropinization. The patient was also started on intravenous immunoglobulin along with steroids. Dramatic improvement was seen in the patient's clinical status, and subsequently, the patient was weaned off from NIV support. Serum AchR antibodies were sent, which came out to be positive, and the patient was clinically diagnosed as a case of Juvenile Myasthenia Gravis with supporting lab evidence. CT chest was done to look for thymus enlargement, which came out to be expected. Now the patient has been shifted to a steroid-sparing agent – Mycophenolate mofetil, along with pyridostigmine, and the patient has shown significant improvement in symptoms.

Conclusion: Myasthenic crisis is a complication of myasthenia gravis characterized by worsening muscle weakness resulting in respiratory failure. Common precipitants of the myasthenic crisis include infections, including aspiration pneumonia, any stress, or any recent change in medication. The outcome of Juvenile myasthenia gravis has improved significantly over the last decade with better recognition, diagnosis, and more effective therapies with good long-term prognosis. Children with JMG exhibit higher rates of remission than adults, including spontaneous remission and remission following a period of drug therapy, with prepubertal children showing the highest rates of spontaneous remission.

Biography

Dr. Ishmeet Kaur Jaggi, is a 2nd-year postgraduate resident in the Dept of Paediatrics at UCMS and GTB Hospital, New Delhi, India. I am extremely interested in Paediatric Neurology and plan to take it up as a super-specialization after my residency. I find keen interest in the marvels of neurology and localization of the lesions in paediatric patients.

Multi-cohort Cerebrospinal Fluid Proteomics Identifies Robust Molecular Signatures for Asymptomatic and Symptomatic Alzheimer's Disease

Muhammad Ali
Washington University in St. Louis, USA

Introduction: Alzheimer's disease (AD), the most common form of dementia, is a complex polygenic disease characterized by the accumulation of Amyloid-β (Aβ42) and hyperphosphorylated Tau 181 (p-tau181) proteins in the brain. An increased level of these proteins, among others, in the AD brains and cerebrospinal fluid (CSF) have been detected years before the symptoms of AD appear. Therefore, studying AD proteome in CSF can reflect its diverse underlying pathophysiology and pave the way for reliable diagnostic and therapeutic advancements. However, a relatively small sample size of existing studies and the use of less throughput protein measurement assays that can only detect a limited range of protein analytes have been significantly hindering the diagnostic and prognostic potential of AD CSF proteome.

Methods: Here, we present one of the largest AD proteomic profiles, based on 7,029 protein analytes measured in CSF of a total of 3,065 individuals in a three-stage study. Firstly, the discovery was performed in 836 samples from the Knight Alzheimer Disease Research Center (Knight ADRC) and 618 samples from the Fundació ACE Alzheimer Center Barcelona (FACE) discovery cohorts using the ATN framework (AT- = 680 and AT+ = 490). Secondly, the proteins that passed multiple test corrections in this dataset were further tested in 832 individuals (AT- = 235 and AT+ = 358) from the Alzheimer's disease neuroimaging initiative (ADNI) and Barcelona-1 replication cohorts. Lastly, the proteins that passed multiple Bonferroni corrections on the meta-analysis were further utilized for creating AD prediction models and conducting pathway enrichment analysis to gain mechanistic insights into AD pathophysiology.

Results: The differential abundance analysis in discovery cohorts identified 3,565 proteins to be significantly (FDR < 0.05) altered. Of these, 2,543 also passed FDR in the replication datasets and had the same direction of effect. Finally, by meta-analysing discovery and replication findings, we identified 2,233 proteins to be significantly (P-Bonferroni < 0.05) altered in AD CSF proteome. Some of the highly significant proteins included YWHAG (P-Bonf < 4.5×10-219), SMOC1 (P-Bonf < 5.8×10-208), PPP3R1 (P-Bonf < 9.2×10-164), NRGN (P-Bonf < 3.2×10-119), and NEFL (P-Bonf < 1.8×10-37). By using lasso regression, we identified a 39 proteins signature with high predictor power (Discovery AUC = 1.0; Replication AUC = 0.99) representing a robust and precise AD diagnostic biomarker. Disease and pathway enrichment analysis highlighted several neurological disorders (e.g., AD, tauopathy, synucleinopathy, and motor neuron disease) and neuronal functions (neuron projection morphogenesis, synapse assembly and organization, axonogenesis, and neuron differentiation) to be significantly enriched in the altered AD CSF proteome.

Conclusion: Our findings show the promising potential of AD CSF proteomics in the development of a reliable and robust AD prediction model. The employed systematic analysis of aptamer-based proteomic data revealed differentially abundant proteins and various biological pathways that are compromised in AD, thereby, increasing our understanding of AD biology. To conclude, our findings may accelerate the development of effective intervention therapies that target the earliest molecular triggers of AD.

Biography

Muhammad Ali is a Postdoctoral Research Associate, Department of Psychiatry, NeuroGenomics and Informatics Center, Washington University in St. Louis, USA. He worked as a Postdoctoral Researcher at Biomedical Data Science, Luxembourg Center for System Biomedicine, University of Luxembourg School for Mental Health and Neuroscience (MHeNS), Maastricht University, Netherlands

Primary Spinal Glioblastoma Multiforme in a Pediatric Patient: A Case Report

Krisverlyn B. Bellosillo*, Myrna Fojas, M.D., PPS, FPNA, Nina M. Alvarez, M.D., Joma Excel G. Bravo, M.D
*Neurology Resident and is the current Chief Resident of the Section of Neurology at Makati Medical Center, , Philippines

Primary spinal glioblastoma multiforme is a sporadic malignant tumor, more so in the pediatric population. Seeding of an intracranial GBM along the spine occurs in 25% of cases, but the reverse process (metastatic intracranial GBM disseminating from a primary intramedullary spinal GBM) is extremely rare. This is a case of a 17-year-old male with a one-month history of progressive weakness and numbness of the bilateral lower extremities, eventually affecting both bowel and bladder control. Neurologic examination showed the last sensory level intact at the T4 dermatome with no sacral sparing, spastic, and hyperreflective bilateral lower extremities with paraparesis, negative bulbocavernosus reflex, and lax anal sphincter tone. Spinal MRI with contrast showed a lobulated expansile T1 and T2 iso to hypointense intramedullary lesion with mild heterogenous enhancement extending from T2 to T6. T3 to T6 laminectomy, laminoplasty, and excision of intramedullary tumor were done. Histopathology and Immunochemistry results were consistent with Glioblastoma multiforme. He completed 28 fractions of radiotherapy (a total of 5040cgy). He had intracranial leptomeningeal metastasis on the 20th month post-operatively. He was started on temozolomide but expired on the 23rd month post-operatively. Due to the rarity of cases, studies on the different therapeutic options are still insufficient. Hence, it remains to have an aggressive course and a poor prognosis despite available management. As recent genetic studies show the difference between pediatric spinal GBM and adult GBM, more studies focusing on the genetic basis of therapeutic options are needed to achieve better outcomes.

Biography

Dr. Krisverlyn B. Bellosillo is a fourth-year Neurology Resident and is the current Chief Resident of the Section of Neurology at Makati Medical Center, Philippines. She finished her Doctor of Medicine at West Visayas State University. She had her Postgraduate Internship at the Philippine General Hospital. During her 3rd year in medical school, she and her teammates won first place in the National Clinico-Pathologic Case Competition.

Current Approaches to the Treatment of Parkinson's: Construction of 3D Neuronal Cell Lines Like the Parkinson Disease Model and Investigation of the Treatment Effects of the Nano Carrier System

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Neurodegenerative diseases have started to attract attention due to their increasing prevalence day by day. Parkinson's Disease (PD) is today's second most common neurodegenerative disease. In the known pathology of Parkinson's disease, the loss of dopaminergic neurons in the substantia nigra of the brain occurs because of the accumulation of the (α)-synuclein component formed by the accumulation of Lewy bodies in specific genes and damaged neurons. The treatment and early diagnosis possibilities of Parkinson's disease, one of the most common neurodegenerative diseases, are minimal. For this reason, the condition needs to be investigated in more detail. There are in vitro and in vivo models in research. However, both types of research have their difficulties. Therefore, new models are needed. In this study, we aimed to develop a 3D culture medium with hydrogel-based bio-ink in neuron cells and then induce Parkinson's disease through neurotoxin. It was aimed to investigate the therapeutic efficacy of the nano-sized exosome-based formulation developed later. Then 2-dimensional (2D) and 3-dimensional (3D) culture mediums were created with the dopaminergic neuroblastoma cell line (SH-SY5Y), and these culture mediums were induced into Parkinson's model. The therapeutic efficacy was investigated by Live & Dead analysis, processing these analysis images in the trainable Weka Program, and finally, by immunostaining method. According to the results, the neuroprotective effect of dopamine-loaded exosomes has been proven in 2D and 3D culture mediums induced in the PD model. At the end of 1 week, the cells were determined to take a spheroidal form. Findings revealed that dopamine-loaded exosomes protect cells against 6-OHDA. Accordingly, we predict that exosome-based treatment methods will be a promising approach to treating Parkinson's.

Biography

She graduated with a Bachelor of Biology in 2013 and a Bachelor of Science in Biology in 2016. In 2022, she graduated from the Department of Bioengineering with a Ph.D. In my Ph.D. thesis. She has also worked as an assistant professor at Altinbas University Health Services Vocational School since 2017. In her academic career, she conducts research on the effects of electroneurophysiological techniques and brain-targeted nanocarrier systems on Parkinson's disease.

Silent Discos for Dementia Care: An Accessible Tool for Music Engagement

Kaylie Glenn*, Anish Ganesh
The Dementia Project, USA

The symptoms of dementia often create a barrier between People Living with Dementia (PLWD) and their care partners, which fuels a cycle of worsening outcomes for both parties. Care partners need a tool that alleviates this communication barrier to improve behavioural outcomes and quality of life. Music is uniquely positioned to fill this void, as memories are rooted rhythmically in the brain in a region labelled the Musical Memory Area within the Auditory Cortex. This region can still be activated and is largely untouched throughout dementia.1 Therefore, effectively engaging to the tune of a familiar song can spark moments of joy, remembrance, and connection.

In response to this growing need for music as an accessible resource, The Dementia Project (TDP) created the Memory Disco, bringing the power of music into group settings such as family gatherings, memory care facilities, and hospitals. The program was designed in collaboration with neurologists from the University of Cincinnati over 8 years, resulting in a multisensory musical experience that combines silent disco technology with our Musical Engagement Program to maximize attention toward the music. TDP is the first organization in the United States to bring silent disco technology to dementia care. Research indicates that silent discos are perceived as useful, and easy to use, and result in positive attitudes in PLWD and care partners alike.4 In fact, silent disco technology targets 3 barriers that limit musical engagement in dementia care: auditory needs, reduced attention span, and limited accessibility to live music. The independent volume adjustment and noise-cancelling nature of each headphone maximize the hearing and attention of each participant. Furthermore, the use of technology allows dementia care partners to bring a powerful engagement tool into any place, at any.

Our Musical Engagement Program adds to the technology component by integrating various engagement techniques to maximize an active listening response. Simply tapping the rhythm on the hands of a participant can lead to a decrease in agitation, an increase in stimulation of sensory systems, and an improved perception of the musical environment around them.5 This can create an organizing effect on movements, emotions, and thoughts, which are crucial to coping with the TDP has conducted over 200 Memory Discos, impacting over 500+ PLWDs and 1,500+ care partners. Its unique ability to engage 15-20 residents with a single facilitator, improve the mood of PLWDs, and increase connection with care partners, makes Memory Discos a prime tool for dementia care. While music is often regarded highly concerning this community, there is a clear barrier to access to the type of music that fosters effective engagement. By leaning on technology to bridge the gap between effective life enrichment and limited resources within dementia care communities, Memory Discos is an innovative tool to combat caregiver burnout and the burden on understaffed facilities. Furthermore, this easily implementable tool introduces the power of music into any place, any time, by anyone– regardless of musical background. As such, this is a vital solution to improve the daily care for the millions affected by the disease today. By understanding the significance of music in dementia care, care partners can utilize music as a tool for building connections and creating new memories in a disease often characterized by what is forgotten.

Biography

Kaylie Glenn (21), B.S. of Neuroscience at Ohio State University, founded The Dementia Project at age 14 as a young violinist. Working in collaboration with neurologists for 8 years at the University of Cincinnati and Ohio State University, her multisensory musical engagement program has involved over 150 young volunteers across Ohio and developed as an innovative solution for all dementia care partners. Kaylie envisions a world where accessible music engagement tools are commonplace in dementia care. As a Community Educator for the Alzheimer's Association, Kaylie is an advocate and supporter for those impacted by dementia.

Effect of a Ganglioside-Containing Drug on Oxidative Processes and PNP Activity in Experimental Autoimmune Encephalomyelitis

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The most common disease of the entire group of myelin diseases is multiple sclerosis. Experimental autoimmune encephalomyelitis in rats is an experimental model of human multiple sclerosis. The purpose of this work is to study the effect of a ganglioside-containing drug on the development of purine nucleoside phosphorylase (PNP) activity in the blood of rats with experimentally induced allergic encephalomyelitis. For studies of oxidative stress, the brain and spinal cord of experimental rats were used. The ganglioside-containing drug Cronassial containing mono-di-tri-sialgangliosides was used as a therapeutic agent.

The experiments were carried out on 30 inbred white rats weighing 180-200 g. Induced immunization with an emulsion of bovine spinal cord homogenate + complete Freund's adjuvant according to the protocol. Rats were divided into groups: a control group; a group with simulated autoimmune encephalomyelitis, and a group with the administering of the drug Cronasial. Animals with EAE were decapitated on the 21st day. Treatment started on the 22nd day. The activity of lipid peroxidation was assessed by the content of hydroperoxides and malondialdehyde. The content of diene and triene conjugates and Schiff bases were recorded by the Deryugina method. The activity of PNP was determined by the accumulation of guanine using the Folin reagent.

Purine nucleoside phosphorylase (PNP, E.C.2.4.2.1) catalyses the cleavage of the glycosidic bond of ribo- and deoxyribonucleosides in the presence of inorganic orthophosphate (Pi) as a second substrate to generate the purine base and ribose (deoxyribose)-1-phosphate.

In the homogenate of the brain and spinal cord of rats on the 21st day of the development of the disease, a stationary level of the intensity of free radical reactions is observed, as well as suppression of the activity of PNP in the blood compared to the animals of the control group. In the animals of the third group, which were administering the ganglioside-containing drug Cronassial, a significant decrease in both initial and final LPO products was observed, as well as an increase in PNP activity. As it is known, PNP is one of the enzymes that characterize the immune status of the organism, and inhibition of this enzyme leads to the disruption of homeostasis of nucleosides that causes T-cell immunodeficiency.

Thus, our data indicate the neuroprotective and antioxidant effects of Cronasial when administered to animals with autoimmune encephalomyelitis.

Biography

Zanginyan Hasmik, scientific worker of the Laboratory of Experimental Biology of the Institute of Molecular Biology of NAS RA. In 2013, she defended her thesis and received her Ph.D. in Biological Sciences. She is the author of more than 40 works published in various journals and conferences.

The Effect of Vitamin B6 on Zebrafish Sociability in a Model for Autism Spectrum Disorder – Preliminary Results

Madalina Andreea Robea
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Usually seen as a complex neurodevelopmental disorder, autism spectrum disorder (ASD) is a long life condition. Characterized by persistent challenges in social communication and interaction, aside from restricted interests and repetitive behavior, ASD has a wide range of risk factors, including environmental agents such as prenatal exposure to different chemicals, drugs, smoking, nutritional factors, or parental issues. Even if it is well-known that genetic predisposition is one of the main risk factors, environmental factors should be investigated due to their salient mode of action. The main aim of the present study was to evaluate the impact of vitamin B6 on an induced ASD-animal model after five days of exposure. For this study, zebrafish was chosen due to its popularity in the research field and also due to its advantages. After the accommodation period ended, zebrafish were randomly divided into four groups: control, vitamin B6 (100 µM), valproic acid (300 µM), and the mixture (100 µM vitamin B6 + 300 VPA µM). Each group had 10 animals. Fish were exposed to the vitamin for one hour, while VPA was constantly present in the experimental tanks.

Before exposure, the initial behavior was registered, followed by the first and last days of exposure. It was tested in the T-maze adapted for the social interaction test, where a social stimulus was placed in the left arm. Each session lasted 4 minutes. Also, heat maps were realized. The data was quantified through the EthoVision software, and the time spent in the left arm was taken to indicate social changes. The results obtained did not reveal significant differences in zebrafish sociability; thus, behavioral trends were recorded for each experimental group. After exposure to vitamin B6, zebrafish presented behavioral improvements compared to the initial behavior.

Regarding the group exposed to VPA, it exhibited decreased time spent next to the social stimulus. When these compounds were given together, zebrafish showed ups and downs for each experimental day. The lack of a more pronounced behavior disturbance may be related to the chosen doses, time of exposure, and mode of exposure. In conclusion, exposure to vitamin B6 and VPA triggered short-term effects in a zebrafish animal model for ASD.

Biography

I am a postdoctoral fellow at the "Alexandru Ioan Cuza" University in Iasi, Romania. My PhD thesis research used animal models, primarily zebrafish, to investigate autism spectrum disorder. Since earning my master's degree, I've been investigating and evaluating the toxicity of certain contaminants connected to human health risks in zebrafish organisms. Because of my deep curiosity in this subject, I was given the opportunity to join two research groups from Iceland and Portugal that helped me gain competence in this field and enhance my professional progress.

Neurogenesis Hypothesis and Clinical Trials of NA-831 for the Treatment of Alzheimer's Disease and Major Depressive Disorder

Lloyd L Tran
Chairman & CSO Biomed Industries, Inc

The hippocampus is critical for learning, and memory contains neural progenitor cells. The hippocampus continues to generate new neurons throughout life, a process known as adult hippocampal neurogenesis (AHN). Hippocampal neurogenesis is persistent through the tenth decade of human life and is detectable in patients with Alzheimer's disease (AD). There was a marked and progressive decline of DCX+ cell numbers in AD patients compared to neurologically healthy individuals. AHN impairment compromises hippocampal function in AD and MCI. This indicates that reduced AHN causes memory impairments and cognitive deterioration in the disease. In addition, AHN is crucial for the regulation of mood. If disturbed, it can have severe consequences for mental health. AHN has been shown to be involved in Major Depressive Disorder (MDD) and Alzheimer's disease pathology. It was observed that AHN impairment takes place before the presence of senile plaques and neurofibrillary tangles (NFTs), both of which are key pathological hallmarks of AD in the dentate gyrus. We proposed that stimulating inherent AHN could be a therapeutic target for improving cognitive function and promoting synaptic resilience. The clinical trials of a new drug, NA-831, presented here provide some evidence that AHN is involved in both AD and MDD,

Phase 2A Clinical Trials of NA-831 for the treatment of Alzheimer's Disease

We reviewed a case study using the results of the Phase 2A study of NA-831 to treat Alzheimer's disease. NA-831 is a small drug molecule that easily crosses the blood-brain barrier with excellent bioavailability. The drug exhibits neuroprotection, neurogenesis, and memory-enhancing properties. A randomized clinical trial of NA-831 was performed on 112 participants with mild and moderate Alzheimer's disease. Half received the drugs, and half received a placebo. The patients with MCI received 10 mg of NA-831 or placebo orally daily. The patients with mild and moderate Alzheimer's disease received 30 mg of NA-831 or placebo orally per day. Subjects with MCI to meet the NIA-AA core clinical criteria for mild cognitive impairment due to Alzheimer's disease. CDR score of 0.5 and a Memory Box score of 0.5 or greater at Screening and Baseline. MMSE score ≥22. Subjects with mild & moderate Alzheimer's disease to meet the NIA-AA core clinical criteria for probable Alzheimer's disease dementia. MMSE: 17-21. NA-831 showed a significant improvement for patients with mild and moderate AD with the ADAS-Cog-13 score change of an average of 4.1 as compared to the placebo after 24 weeks of treatment ($p = 0.001$; ITT). CIBIC-Plus showed 78 % of patients improved ($p = 0.01$; ITT). mNA831 was well-tolerated at 30 mg/day. There were no serious adverse events observed.

Phase 1B Clinical Trials of NA-831 for Major Depressive Disorder (MDD)

We completed a Phase 1B pilot study, which was a randomized, double-blind, fixed-dose, placebo-controlled, active reference study to investigate the efficacy, safety, and tolerability of two fixed doses (20 and 40 mg/d) of NA-831 vs. that of placebo after 6-week treatment in 32 adult patients with major depressive disorder (MDD). Venlafaxine XR was used as the active reference. The most common adverse effects reported in the functional NA-831 treatment groups were mild headache and dry mouth. Both doses of NA-831 resulted in a significant improvement compared to placebo on the primary efficacy analysis. The difference between active treatment and placebo of 7 points on the MADRS translates into a clinically relevant difference in response rates of 32.5 % units, compared to an average of 16% units for antidepressants approved by the USA and European health authorities. Treatment with NA-831 for 6 weeks was well tolerated and efficacious in reducing depressive and anxious symptoms in patients with MDD. Conclusion: The Neurogenesis Hypothesis has been shown to be a viable approach for further research for Alzheimer's disease (AD) and Major Depressive Disorder (MDD). Biomed is in the process of conducting Phase 2B and Phase 3 trials of NA-831 for the treatment of AD and MDD.

Biography

Lloyd is a scientist with 25 years of experience in drug development and clinical trial management. He is an inventor with many patents in drug therapeutics to treat neurological and infectious diseases. Lloyd serves as the chairman and CSO of Biomed Industries, Inc., the parent company of Biomed Pharmaceuticals, NeuroActiva, Biomed AI, and MedAware Systems, Inc. Biomed is conducting phase 2/3 of NA-831 for the prevention and treatment of Alzheimer's Disease. In his early career, he was employed as a research scientist at G.D. Searle (a subsidiary of Pfizer), and was the director of R&D at Biomed Pharmaceuticals. Lloyd graduated with a BSc(Honours) and completed a PhD in medicinal chemistry at the University of Otago and Wellington University of New Zealand.

Anti-oxidative Effect of Ginkgolide B on iNOS and NO in Rat Primary Microglia Induced by LPS

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Background: Ginkgolide B (GB) has been shown to have a neuroprotective effect on ischemic stroke, neurons against ischemia-induced apoptosis, white matter lesion caused by chronic cerebral hypoperfusion or NLRP3 inflammasome in the activated BV2 cells induced by Aβ1-42. However, it still remains unknown if Ginkgolide B can modulate the oxidative stress in the microglial activation induced by Lipopolysaccharide (LPS).

Objective: To know the effect of GB on microglial activation, like inducible nitric oxidative synthase (iNOS) and nitric oxide (NO)upregulation by LPS.

Method: The primary microglia were obtained from the primary mixed-glial cultures, which were derived from 2-3d SD rats, after 10-day growth using the shake-off method. The primary microglia were seeded into the 24-well plates at the density of 3.5 x105 cells/well while BV2 were seeded at the density of 1x 105 cells /well, and then they were incubated at 5% CO2, 37 for 24h before any addition. Based on the experience in our previous exploration of GB on oxidative stress in the mixed glia, 10μM of Ginkgolide B was used to the activated microglia induced by 100ng/ml of LPS in this study. Additionally, SB inhibitors related to MAPK signaling pathways as exploring working mechanisms or Aspirin as a positive control were enrolled into in BV2 experiments. The cells or supernatant were harvested after co-incubation for 12h, 24h, or 36h. The production of nitric oxide in the supernatant released via microglia was detected by Griess assay, and iNOS expression in the cells was measured by immunofluorescent antibody.

Result: The results showed that both primary microglia or BV2 were activated by 100ng/ml of LPS at 12h, 24h, or 36h, which included the upregulation of NO and iNOS in a time-dependent manner. When GB was added into the cells for 12h, 24h, or 36h, NO and iNOS were down-regulated, which showed a time-dependent manner. Compared to LPS, NO value down-regulation by GB was significant (P0.05 at 24h- or 36h-treatment of GB). Interestingly, we measured some inflammatory molecules such as COX-2 or TNF-α in cell lysates at the same time points by Western Blotting analysis and found that GB could down-regulate the inflammatory response induced by LPS as well.

Conclusion: Ginkgolide B could down-regulate the oxidative stress in not only the primary mixed glia but also in the primary rat microglia or mouse microglial cell line BV2 induced by LPS, which suggested that GB potentially has pharmacological value in intervention to CNS disorders with oxidative stress and inflammation. This study provided more evidence to use GB in the therapy at the early stage of neurodegenerative diseases, including Alzheimer's disease or PD.

Biography

Ling Guo, M.D. Prof., Director. P.I. awarded grants: total 10. Key grants: (1) National Natural Science Foundation of China, 340000RMB, No. 81760228. (2) Kunming Institute of Research on Neurodegenerative diseases. 300000RMB, No.2020-SW-30. (3) Yunnan Scientific Technology Plan for Talent and Platform "Yunnan Station of He Zhendan Academician," Rank#2, 3600000RMB No.202305AF150139. Training and Career: (1) Physician, Dept. of Internal Medicine, The 2nd Affiliated Hospital, Kunming Medical Univ. 1983.2.-1993.8. (2) Associate Professor, Internal Medicine, The 2nd Affiliated Hospital, Yunnan, China. (3) Postdoctoral fellow to Senior Research Associate 1998.2.-2011.12.31. Dept. Of Cell & Molecular Biology-- Neurology &Alzheimer's Center, Medical sch. Northwestern Univ., USA. Projects: (1) Study on Neuroinflammatory Mechanisms of Alzheimer's disease. (2) Drug discovery. Publications: 55. Patent: 3 @rank#1.

Line Quadrisection Test as a Diagnostic Tool for Assessing Spatial Awareness in Alzheimer's Disease Dementia Patients

Yurim Jin
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Alzheimer's disease (AD) is a neurodegenerative disease affecting 50 million people worldwide, characterized by brain atrophy, memory loss, and cognitive decline. While much has been researched about the neuropathology and cognitive decline of AD, the motor symptoms received less attention. Previous studies that have conducted the Line Bisection Test (LBT) on AD patients failed to identify abnormal results. However, some studies have added measures to induce an attentional bias in the participant, such as the character line bisection task and optokinetic stimulation. So, in this study, I aimed to determine if AD patients' bilateral temporoparietal damage can be detected through the Line Quadrisection Test (LQT), a novel test modified from the Line Bisection Test (LBT). In the LQT, participants must mark a vertical line's ¼, ½, and ¾ positions. I conducted the LQT on 50 Alzheimer's disease dementia (ADD) patients and 50 normal controls (NC). When marking the ¼ and ¾ points, ADD patients deviated more laterally than normal people but showed normal results on the bisection task (1/2 point). When quadrisecting the lines, the participants' attentional focus shifts toward each end, and the reduced spatial awareness of ADD patients causes a more drastic deviation. Thus, this study reveals that LQT detects the spatial abnormalities of ADD patients that weren't noticed in the LBT and can be used as a simple but effective tool to diagnose AD in advance. While current diagnostic measures like MRI and PET are too costly and cognitive tests are lengthy and burdensome, the LQT can indicate the possibility of AD in a faster and more affordable way. Furthermore, the LQT can be made in a digital app format, enabling people to conduct the test independently regardless of time and place, contributing to wider access to pre-diagnostic measures for AD. Thus, this study not only illuminates a novel aspect of an abnormal motor symptom of AD but also has valuable implications for wider access in pre-diagnosing AD.

Biography

Yurim Jin is a neuroscience researcher at the Korean Minjok Leadership Academy, in South Korea. Head of the research department of Korean Youth Society for Neuroscience (KYSN).

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