magnus Group

6TH EDITION OF INTERNATIONAL CONFERENCE ON

NEUROLOGY AND BRAIN DISORDERS

OCTOBER 24-26, 2022 | ORLANDO, FLORIDA, USA

Venue:

Hilton Garden Inn Lake Buena Vista/Orlando
11400 Marbella Palm Ct, Orlando, FL 32836, United States

BOOK OF ABSTRACTS

INBC 2022

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24-26 OCT

BOOK OF
ABSTRACTS

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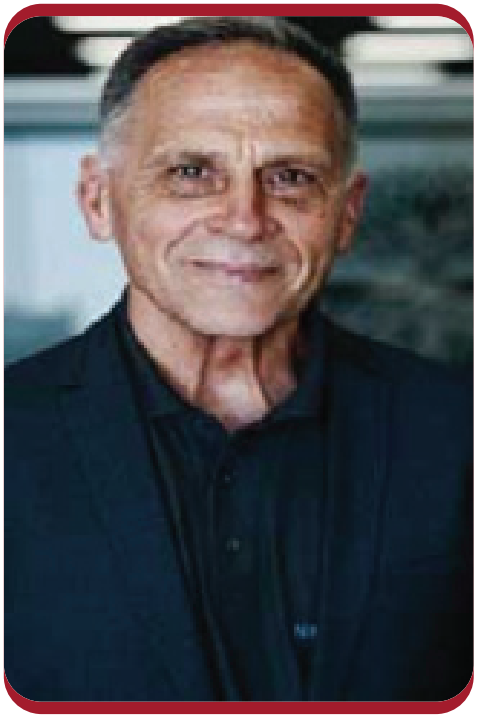


Dear Colleagues and all Attendees of INBC 22,

On behalf of the distinguished members of the Scientific Committee for the 6th Edition of the 'International Conference on Neurology and Brain Disorders and the dynamic organizing team behind this event, it is my pleasure and honour to welcome you all to INBC 2022. Each year, since the inaugural 1st INBC, this conference has proven to provide a highly generous offering of a very diverse range of distinguished speakers presenting their innovative research, interventions and therapies. Many collaborations have been formed from past conferences and the sharing of ideas and research findings has cross inspired every attendee at these conferences in some manner. INBC 22 has shaped up to be one of the most outstanding conferences of the year in this field of interest and research. I wish you all every success with your INBC 22 presentations and great enjoyment and inspiration when attending other speakers' presentations.

Ken Ware
NeuroPhysics Therapy Institute, Australia

Welcome Message

Dear congress visitors,

COVID-19 stopped our normal way of life for several years. Did neurological disorders stop occurring during COVID-19 ? Did people stop getting stroke ? How about meningitis ? Alzheimer's disease and dementia ? No. In fact, many brain disorders became worse as patients lost access to traditional healthcare and resources that were directed for COVID-19. Mental health issues, which are closely related to physical health issues, also deteriorated during COVID-19 causing for increased concerns for neurological disorders. Neurological research even slowed during COVID-19 as Universities and Medical Centers shut down labs (unless you were working on COVID-19) and pharmaceutical companies directed attention and money elsewhere. It is more important now than ever to make up for lost time. Now that in-person conferences have resumed, it is clear we have a lot of ground to make up !

This conference will provide an excellent opportunity for us to discuss and debate neurological research, and I strongly encourage you to attend and actively participate. All of us in the scientific community need to take advantage of every opportunity to discuss the future of neurological disease prevention, diagnosis, and treatment. It is only through active participation that we can get back on track and help our patients in most dire need.

I hope to see you in Orlando where we can make up for lost time !

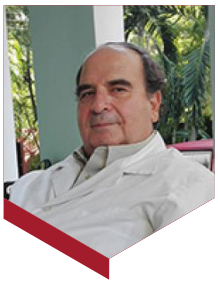
Thomas J. Webster, Ph.D.
Interstellar Therapeutics, United States

Keynote Speakers

Ken Ware
NeuroPhysics Therapy Institute, Australia

Thomas J. Webster
Interstellar Therapeutics, United States

Andreas Till
University of Bonn, Germany



Calixto Machado
Institute of Neurology and Neurosurgery, Cuba

W S El Masri
Keele University, United Kingdom

Roy G Beran
Western Sydney University, Australia

Torbjorn Backstrom
University of Umea, Sweden

Thank You All...

ABOUT
MAGNUS GROUP

Magnus Group (MG) is initiated to meet a need and to pursue collective goals of the scientific community specifically focusing in the field of Sciences, Engineering and technology to endorse exchanging of the ideas & knowledge which facilitate the collaboration between the scientists, academicians and researchers of same field or interdisciplinary research. Magnus Group is proficient in organizing conferences, meetings, seminars and workshops with the ingenious and peerless speakers throughout the world providing you and your organization with broad range of networking opportunities to globalize your research and create your own identity. Our conferences and workshops can be well titled as 'ocean of knowledge' where you can sail your boat and pick the pearls, leading the way for innovative research and strategies empowering the strength by overwhelming the complications associated with in the respective fields.

Participation from 90 different countries and 1090 different Universities have contributed to the success of our conferences. Our first International Conference was organized on Oncology and Radiology (ICOR) in Dubai, UAE. Our conferences usually run for 2-3 days completely covering Keynote & Oral sessions along with workshops and poster presentations. Our organization runs promptly with dedicated and proficient employees' managing different conferences throughout the world, without compromising service and quality.

ABOUT
INBC 2022

Neurology conference organized by Magnus Group has been the preeminent global conference for neurology professionals over the last 5 years and we take great pride in organising and inviting all the global delegates, to unite once again at the "6th Edition of International Conference on Neurology and Brain Disorders" (INBC 2022).

The conference will enlighten the theme "Exploring the Grey Areas of Neurological Research" and will focus on its agenda to bring together distinguished scientists, researchers, academicians, notable neurologist, healthcare professionals, surgeons, nurses, caregivers, pharmacists, industry icons and government representatives to discuss the latest innovations, trends, and problems in the field.

The introduction of fresh technical capabilities has spurred the evolution of neuroscience, and the rate at which these capabilities are being developed has accelerated substantially in the last decade. The objective of this conference is to generate innovative treatment ideas that will benefit individuals with a variety of brain disorders.

Through its keynote sessions, oral and poster presentations the symposium will feature cutting-edge research in every subspecialty of neurology and brain disorders whilst providing the chance to reconnect with peers from around the world. The major goal is to raise awareness about adequate mental health care, the risks associated with improper treatment, and the impact on mental health patients, as well as to discover cure options for Brain Disorder.

BOOK OF ABSTRACTS

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DAY 01

KEYNOTE FORUM

6TH EDITION OF

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Nanomedicine and the brain: A perfect combination?

Nanotechnology has revolutionized numerous industries, particularly medicine. Nanoparticles, nanotubes, drug delivery nanoparticles and nanotextured neural probes are just several of many examples where nanomedicine is positively impacting neuroscience. This presentation will summarize some of the more prominent studies where nanomedicine has been used to treat various neurological disorders. Specifically, it will cover in vivo studies which have demonstrated a faster and greater return of motor function to stroke induced rats when implanted with stem cells and carbon nanotubes. Further, it will cover the design of nanoparticles that can penetrate the blood brain barrier to more effective deliver drugs to treat Parkinson's disease. New self-assembled nanomaterials will also be covered which can kill bacteria in the brain. This presentation will also present what studies are needed for the field of nanomedicine to continue to positively impact neurological diseases.

Audience Take Away:

- How nanomedicine is being used to treat a wide range of neurological disorders
- Examples of nanomaterials and their attractive properties for medicine
- Disadvantages of nanomaterials and their use in the brain as well as what future directions are needed for the field to grow

Thomas J. Webster

Interstellar Therapeutics,
Boston, MA, USA

Biography

Thomas J. Webster's (H index: 109; Google Scholar) degrees are in chemical engineering from the University of Pittsburgh (B.S., 1995; USA) and in biomedical engineering from RPI (Ph.D., 2000; USA). He has served as a professor at Purdue (2000-2005), Brown (2005-2012), and Northeastern (2012-2021; serving as Chemical Engineering Department Chair from 2012 - 2019) Universities and has formed over a dozen companies who have numerous FDA approved medical products currently improving human health. Dr. Webster has numerous awards including: 2020, World Top 2% Scientist by Citations (PLOS); 2020, SCOPUS Highly Cited Research (Top 1% Materials Science and Mixed Fields); 2021, Clarivate Top 0.1% Most Influential Researchers (Pharmacology and Toxicology); and is a fellow of over 8 societies.

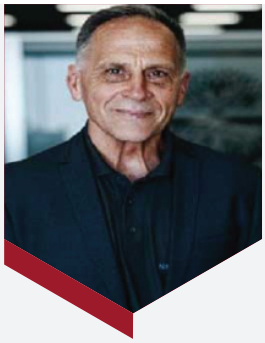
Rogue percepts= rogue responses: What are the possible sets of initial conditions that give rise to willis-ekbom disease - Restless Legs Syndrome (RLS) and can RLS be therapeutically pacified? A neurophysics therapy study and hypothesis based upon observations and outcomes

The human Central Nervous System (CNS) fundamentally does something elegant and simple and is programmed to abide by some simple sets of rules and initial conditions. Fundamentally, the CNS takes in and perceives information coming in from the outside world and responds to it. However, the keyword here is 'perceives'. Human perceptions of same environmental information may vary significantly from one individual to the next and therefore psychophysical responses may also vary significantly between numbers of individuals who are all having the same environmental experience as each other. The Onset, Incubation and Manifestation (OIM) of Psychophysical Disease and Disorder (PDD) involves, perception action and cognition. Through this lens we investigate the sets of initial conditions that could be playing a strong role in the OIM of willis-ekbom disease - Restless Legs Syndrome (RLS) a condition that affect at least 5% of the general population. And can RLS be therapeutically pacified?

This presentation will include case studies of patients who presented with chronic RLS and who were able to systematically calibrate their CNS and accomplish pacification of their RLS in a very small time frame via NeuroPhysics Therapy.

Note: Whilst RLS has been selected as the concerning psychophysical condition for this keynote presentation, the perception, action and cognitions rules are the same for the OIM of all psychophysical diseases and disorders, many of which are reaching almost epidemic proportions, with most people who are suffering from a PDD having no family genetic history to refer to as a root cause. Besides, referring to genetic inheritance as a root cause for the OIM of psychophysical diseases and disorders can only reach so far as there is no reason to speculate that a person will become a world champion athlete simply because one of their parents were. Genetic inheritance should work across all human conditions, good, bad or indifferent and we know that this is not the case.

Keywords: Central Nervous System, Environment, Restless Legs Syndrome (RLS), Perception Action & Cognition, Psychophysical Diseases and Disorders, Sets of Initial Conditions, Calibrate, NeuroPhysics Therapy.



Ken Ware

Department of Research and Development, NeuroPhysics Therapy Institute, Gold Coast, Qld, Australia

Biography

Ken Ware was founder of Neurotricional Sciences Pty Ltd and NeuroPhysics Therapy and Research and he had been in private practice for almost 30 years, while doing independent and collaborative research. He also presented unique research at 10 major International Science Conferences including neuroscience, Physics, Psychology and Life Sciences, which covers a very broad scientific audience. He is Former Mr. Universe 1994, National powerlifting and Bodybuilding champion and record holder. He had published relative publications in 'Frontiers in Clinical Physiology' - 'World Journal of Neuroscience' – 'World Journal of Cardiovascular diseases'. He is recipient of Her Majesty, Queen Elizabeth's' 'Australian Sports Medal' - in 2000, in recognition for personal contributions to the development of the Australian Sporting Culture.

In vitro modelling of brain development and neurodegenerative diseases

Given the complex etiopathology of neurodegenerative diseases and the limited access to vital human brain cells as suitable research platform, there is an increasing demand of innovative human-specific model systems that help elucidating the causative mechanisms of these disorder and open the avenues for novel prevention and/or treatment strategies. The advent of cell reprogramming has enabled the generation of induced Pluripotent Stem Cells (iPSCs) from patient fibroblasts or blood cells and their subsequent differentiation into tissue-specific cells, including neurons and glia. This approach in combination with in vitro genome-editing technology is suitable to recapitulate disease-specific phenotypes but also neurodevelopmental aspects in classical cell culture paradigms and thus represents an invaluable asset for developmental research, disease modelling and drug validation in the framework of personalized medicine.

Here we demonstrate how in vitro modelling of neurodevelopment and neurodegeneration in classical two-dimensional and 3D model systems may unveil underlying causative mechanisms and target pathways. First, we demonstrate that the cytoprotective NRF2 signaling pathway is upregulated and activated during neuronal differentiation from neural stem cells to maturing neural populations. We continue to show how this pathway is regulated and able to protect neural populations from environmental insults, e.g. oxidative stress, during development. Next, using iPSC-derived dopaminergic neurons from a Parkinson's Disease (PD) patient carrying the causative LRRK2 G2019S mutation, we demonstrate that mutant carrier cells display defects in homeostatic turnover of mitochondria ('mitophagy') as early as at day 8 of differentiation. These data pinpoint one important causative mechanism in the pathology of PD, i.e. quality control of mitochondria, and emphasize the vital role of mitophagy regulation during early steps of neuronal development. Moreover, we asked which subcellular pathways contribute to formation of Neuronal Intranuclear Inclusions (NIIs) of mutant Ataxin-3 protein in an iPSC model of polyglutamine disease (spinocerebellar ataxia type 3/SCA3). Inhibition of autophagy (an essential lysosomal degradation system) resulted in a significant increase of NIIs in the susceptible cell population, and the build-up of NIIs could be abated by pharmacological upregulation of autophagic flux. As outlook, we present our approach on how to dissect the connection between diet, the gut microbiome composition, brain circuitry and manifestation of metabolic diseases such as obesity and its comorbidities (including dementia) using a translational approach in combination with in vitro model systems.

In summary, our work emphasizes the usability of innovative in vitro models for assessment of development, disease pathways and even organ crosstalk.

Andreas Till*, Johannes Jungverdorben, Beatrice Weykopf, Vesselina Semkova, Lea Flitsch, Wiebke K. Fenske, Oliver Brustle

University Hospital of the University of Bonn, Bonn, Germany

Biography

Andreas Till is a dedicated cell biologist whose research focuses on cellular stress pathways associated with human diseases. He received his PhD at the University of Kiel, Germany, where he studied immune signaling and pathogen detection. After receiving a research fellowship, he worked at the University of California San Diego where he focused on selective autophagy. Next, he joined the University of Bonn as lecturer for Molecular Biomedicine, and was later appointed lab head in the Section for Metabolic Diseases. Over the last years, he has specialized on in vitro cell models for stress pathways such as antioxidative responses, autophagy and metabolic reprogramming. Recent projects aim to elucidate the interconnection between the gut microbiome and neural circuits regulating energy homeostasis in obesity. Dr. Till has published more than 60 papers and presented his research at several international conferences in Europe, USA, Japan and China.

Audience Take Away:

- which in vitro model systems are nowadays available to study disease mechanisms in the dish
- which histological and molecular features could be used in the clinic to interpret pathological findings
- which shared cellular and molecular events underly the pathogenesis of neurodegenerative diseases
- How preclinical drug screening can be performed on authentic human cells

The case of jahi mcmath: Dilemmas and controversies in the diagnosis of brain death

I was a main expert in the case of Jahi McMath, who was diagnosed with Brain Death (BD). Nonetheless, ancillary tests performed nine months after the initial brain insult showed conservation of intracranial structures, EEG activity, and autonomic reactivity to the "Mother Talks" stimulus. She was clinically unarousable and unresponsive, without evidence of self-awareness or awareness of the environment. However, the total absence of brainstem reflexes and partial responsiveness rejected the possibility of a coma. Jahi did not have Unresponsive Wakefulness Syndrome (UWS) because she was not in a wakefulness state and showed partial responsiveness. She could not be classified as a Locked-In Syndrome (LIS) patient either because LIS patients are wakefuland aware and although quadriplegic, they fully or partially preserve brainstem reflexes, vertical eye movements, or blinking, and respire on their own. She was not in a Minimally Conscious State (MCS) because she did not preserve arousal and preserved awareness only partially. The CRS-R resulted in a very low score, incompatible with MCS patients. MCS patients fully or partially preserve brainstem reflexes and usually breathe independently. MCS has always been described as a transitional state between a coma and UWS, but it has never been reported in patients with all clinical BD findings. This case does not contradict the concept of BD but brings the need to use ancillary tests in BD again for discussion. I concluded that Jahi represented a new disorder of consciousness, non-previously described, which I have termed "Responsive Unawakefulness Syndrome" (RUS).

Calixto Machado*, MD, PhD, FAAN

Institute of Neurology and Neurosurgery, Havana, Cuba

Biography

Dr. Calixto Machado graduated as MD in 1976. He continued his medical training to become a Specialist in Neurology and Clinical Neurophysiology, First Degree in 1980 and Second Degree in 1984. In 1990 he became the youngest Dr. in Sciences (Second Degree) in his country. Dr. Machado received clinical training in specialized neurology centers in Sweden, Italy, Austria, and the US. Dr. Machado has trained many medical students, neurology residents, fellows, and doctorate students in neurosciences from Cuba and the rest of the World. During the last 30 years, he has run many research protocols, such as brain death, coma, persistent vegetative states, and other disorders of consciousness, stroke, autism, autonomic nervous system, COVID-19, among others.

He joined Cuba to the WFN, and it appears within the Latin American Region with the name "Institute of Neurology and Neurosurgery." For many years I have attended the WFN conferences representing his country. He has over 600 peer-reviewed articles, book chapters, and seven books. His Book "Brain Death: A Reappraisal" has been recognized with great enthusiasm

among neurologists, neurosurgeons, intensivists, and physicians specializing in transplants. He has organized eight International Symposia on Brain Death and Disorders of Consciousness in Havana since 1992, welcoming the main worldwide experts in this area. This is probably the Conference with more continuance in time in the World. In 1992, he was the first Cuban neurologist who was a member of the American Academy of Neurology (AAN), nominated as a Corresponding Fellow. He has been the President of the Organizing Committee of eight International Symposia on Brain Death and Disorders of Consciousness since the early '90s. During these symposia, he has organized many continuing education courses in neurology attended by Cuban, Latin-American, and US young neurologists.

He is the principal Professor of Neurology and Clinical Neurophysiology in this country. He has been the mentor of 57 physicians running their residency programs to become neurologists and 18 Ph.D. aspirants, many of them from Latin-American countries and the US. He has been awarded 25 times with the "Best Annual Scientific Medical Research in Cuba" and five years with the "Cuban Academy of Science Annual Award." In 2005 he was bestowed with the American Academy of Neurology "Lawrence McHenry Award." This was the first time that a Hispanic neurologist, and a neuroscientist from a developing country, received this recognition. In 2011, he was awarded as the "Researcher of Year" by the International Academy for Child Brain Development (Philadelphia, USA) and by the International Association of Functional Neurology and Rehabilitation (Orlando, USA). He is a Senior Professor and Researcher in neurology and clinical neurophysiology at the Institute of Neurology and Neurosurgery, Havana, Cuba, and the President of the Cuban Society of Clinical Neurophysiology and the National Commission for the Determination of Death; he chaired the edition of the Cuban Law to diagnose death.

Dr. Machado is a Corresponding Fellow of the American Academy of Neurology, Chairman of the Network on Defining Death of the International Association of Bioethics, member of The World Federation of Neurology and the International League against Epilepsy. He is a Senior Academics of the Cuban Academy of Sciences. In the academic field, Dr. Machado is recognized as a world expert in neurological disorders such as brain death, coma, disorders of consciousness, neuroimaging, clinical neurophysiology, stroke, and recently on the way SARS-CoV-2 attacks the nervous system. He was the main neurological expert in the Jahi McMath case. His Faculty appointments as visiting professor include Miami University (Department of Neurology, Department of Philosophy and Bioethics), Memorial Sloan-Kettering Cancer and Cornell University in New York, the Institute of Neurology at Columbia University, the Neuroanesthesia and Neurocritical Care Service at Johns Hopkins Hospital, the George Washington University, and the Department of Neurology and Neurosurgery at Jackson's Memorial Center in Miami. During the last 15 years, Dr. Machado has been invited to Johns Hopkins as a key-note speaker, and he has impressed the Neuroscience Critical Care team, stimulating scholars to begin a fruitful scientific exchange with Dr. Machado. An International Visiting Scholarship Program has supported him, co-sponsored by Johns Hopkins University and a private foundation. He has been consulted as Expert Advisor for contentious suspected brain-dead cases in different countries, such as the Jahi McMath case in the USA, and to treat and diagnose comatose and unresponsive wakefulness syndrome patients (Spain, Mexico, Argentina, Brasil, USA, Saudi Arabia).

Prognostic indicators of spontaneous neurological recovery following traumatic spinal cord injuries and the role of the neurologist

Spontaneous Neurological Recovery is very common in patients with incomplete traumatic spinal cord injuries. This is provided no further mechanical and non-mechanical damage is inflicted onto the injured cord. Currently there is great deal of emphasis on the threats of the Biomechanical Instability of the spinal axis, canal encroachment and cord compression being a source of further neurological damage or preventing neurological recovery. What is not currently highlighted is that the acutely injured Spinal Cord is also Physiologically Unstable and unable to defend itself from systemic complications such as severe hypoxia, hypotension, hypothermia, generalised sepsis and electrolyte imbalance which can equally cause neurological deterioration, delay or absence of neurological recovery if the patient is inadequately managed. These complications would not normally damage an intact spinal cord as well as from mechanical damage.

Neurological recovery has been observed to occur With Active Physiological Conservative Management (APCM) of both the injured spine and the multisystem effects of the cord damage since the 1950s. This observation is still being repeatedly confirmed for more than half a century in patients presenting early, with any long tract sparing, irrespective of the severity of the radiological presentation of the spinal injury on Xrays, CT or MR and without intervention on the spine or spinal cord

The last two decades have witnessed an aggressive promotion of early interventions, surgical and non-surgical within a window of opportunity which, following a post-hoc analysis, has been extended from 4 hours in the laboratory animal to 24 hours in humans. Assertions are increasingly being made that surgical decompression of the spinal canal within 24 hours of injury results in a better neurological outcomes than with late surgery and must therefore give better result than APCM. These claims were facilitated by a change of the established neuro-functional outcome assessment tool to a numerical tool conveying the statistical shift of patients from defined classes to new classes based on the exhibited motor power by the patient in the first 24 hours of injury. The accuracy of such an early numerical assessment and documentation of motor power in newly injured , anxious, paralysed patients, in pain, under heavy analgesic medication and sedation , with possible associated injuries has yet to be validated for its reliability as a baseline for subsequent comparison of outcomes of interventions.

The positive and negative prognostic indicators of neurological recovery, and the factors that enhance, prevent or cause neurological deterioration in patients with complete and incomplete cord damage will be discussed.

W S El Masri MB, BCh, FRCS, FRCP

Hon. Clinical Professor of Spinal Injuries – Keele University
Emeritus Consultant Surgeon in Spinal Injuries

Robert Jones & Agnes Hunt Orthopaedic Hospital - Oswestry
Past President of International Spinal Cord Society (ISCoS)

Trustee of the Institute of Orthopaedic RJAH Hospital Oswestry SY10 7AG Chairman of Trustees of SPIRIT Educational Charity

Biography

Prof W S El Masri FRCS Ed, FRCP currently hon. clinical professor of Spinal Injuries (SI), Keele University has trained between 1971 & 1983 in the Oxford Group of hospitals, Guys & Stoke Mandeville hospitals and the USA. He obtained the first accreditation in Spinal Injuries and General Surgery in 1982. Appointed consultant surgeon in spinal injuries at the Midland Centre for Spinal Injuries in 1983. He personally treated 10,000 patients with. He published 145 manuscripts. He the author of the: Concepts of "Physiological Instability of the Spinal Cord", "Time related Biomechanical Instability", "Micro-instability of the injured spine" and published the largest series of Bladder cancer in SCI patients. He has repeatedly demonstrated and published on the discrepancy between the radiological and neurological presentation

The influence of the initial force of the impact that damages the neural tissues, the severity of the cord damage and the quality of care of the injured spine and its consequent systemic effects will be discussed.

To date there is no evidence that surgical or other interventions on the injured spine and/or spinal cord add value to the neurological or other outcomes of patients with TSCI.

Further mechanical and non-mechanical damage to the injured spinal cord during the early stages of TSCI remain the main causes of neurological deterioration, delays or lack of expected recovery following TSCIs.

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 world-wide. He won many National and International awards.

BOOK OF ABSTRACTS

INBC 2022

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DAY 01

SPEAKERS

6TH EDITION OF

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NEUROLOGY AND BRAIN DISORDERS

IN-PERSON

Fulya Turker*[1], Seth S. Margolis[1,2]

[1]Department of Biological Chemistry, The Johns Hopkins University School of Medicine, Baltimore, MD

[2]Solomon H. Snyder Department of Neuroscience, The Johns Hopkins University School of Medicine, Baltimore, MD

Application of activity-based probe, MV151, in the mammalian nervous system reveals new insight into proteasome changes in human alzheimer's disease brain

Proteasome complexes play a critical role in human brain health and disease. Despite enormous effort, a deep understanding of proteasome composition, activity and abundance in the human brain remains poorly understood. Here, we describe a series of tools that can measure the catalytic activity and levels of individual proteasome □-subunits and distinct proteasome complexes in mammalian tissue. Adapting these tools to brain tissue from human Alzheimer's Disease (AD) patients, we found that the human brain has a high abundance of catalytically active 20S proteasomes and that these 20S proteasomes exhibit higher total activity in AD when compared to unaffected controls. Additionally, we showed that 20S proteasome abundance is inversely correlated with the severity of the AD case. Taken together, these data indicate that while 20S proteasome abundance is decreasing in AD patient brains, this is counter-balanced by an increase in proteasome activity. We now propose that homeostasis of 20S proteasome activity is a hallmark of human brain health, and changes in abundance and activity are correlated to the severity of AD disease. This discovery sets the stage for further investigation into 20S proteasome activity in neurodegenerative disease states.

Audience Take Away:

- He will present a novel application of multiple proteasome tools to study the proteasome composition, abundance, and activity using purified proteasome, mouse brain tissues, and neuronal cultures to inform our efforts to study proteasome biology in human AD patient brains
- The audience will learn how to adapt this new approach to studying proteasome biology in different model organisms and across different tissues/disease states
- Studying proteasome activity in postmortem human brain tissue has been very challenging. The audience will be able to incorporate this new approach to their studies to study protein degradation in human tissue samples
- One of the hallmarks of Neurodegenerative Diseases (NDs) is the impairment of proteasome-dependent protein degradation pathways. The link between proteostasis and ND progression is an active area of investigation. The audience will be able to design experiments with the activity-based probe used in this study to investigate proteasome activity in various NDs (such as AD, HD, PD, etc.)

Biography

Fulya Turker studied Molecular Biology and Genetics at the Sabanci University, Turkey, and graduated with a BS in 2017. She then joined the research group of Prof. Seth S. Margolis at the Department of Biological Chemistry, Johns Hopkins University School of Medicine. She is in her final year of PhD study on protein degradation in the nervous system.

IN-PERSON

Arman Fijany*[2], Nadeem Al-Adli[2], Darryl Narcisse[3], Robert Benkowski[1], Ashutosh Tripathy[3], Samarendra Mohanty[4]

[1]Designplex Biomedical, Fort Worth, TX, USA
[2]Texas Christian University School of Medicine, Fort Worth, TX, USA
[3]Opsin Biotherapeutics Bedford, TX, USA
[4]Nanoscope Technologies Arlington, TX, USA

Multi-characteristic opsin gene delivery for neuromodulation of inflammatory and persistent pain sensation in rodent model

Pain is an uncomfortable sensation perceived by the nervous system that can have a variety of different causes. Pain can be seen as a complication of disease, emotional states and even as a standalone pathology. The neuroscience of pain is poorly understood. Multiple levels of complex neurological signals are thought to be involved in pain perception and modulation. Treatments for pain symptoms are limited and can be associated with severe and even potentially deadly, side effects. The Anterior Cingulate Cortex (ACC) within the corpus callosum and the Dorsal Root Ganglion (DRG) of the spine are two regions thought to be involved in pain neural pathways and a potential target for neuromodulation. We created a highly sensitive optogenetic actuator Multi-Characteristic Opsin (MCO), which is expressed and targeted to inhibitory (GABAergic) neurons via a Glutamic Acid Decarboxylase promoter. This opsin was delivered to a mouse model through both viral and laser gene delivery methods. With the use of a bluetooth stimulator device, we provided red light stimulation (630 nm) of these inhibitory neurons within the ACC and DRG at multiple stimulation frequencies and intensities. Pain responses and modulation were then investigated via a formalin challenge and sciatic cuff induced neuropathy, which mimicked acute inflammatory and persistent neuropathic models, respectively. We then measured pain responses via standard pain response behaviors such as hind paw lifting and licking. Chronic pain responses were also monitored via preference for cage regions that provided increased light stimulation associated with pain relief. Our results demonstrated that mice that expressed the optogenetic MCO had a significantly decreased pain response in response to both acute and chronic pain assays when subject to red light stimulation. More specifically, most of the decrease of reflexive pain was seen in the delayed, inflammatory phase that occurred around 20 minutes after formalin injection. In the control mice groups that did not express the optogenetic MCO and in the mice that expressed the optogenetic MCO but were not stimulated by red light, there was no significant difference between baseline reflexive pain values. Taken together, the delivery of the optogenetic MCO, coupled with direct red light stimulation of the DRG and ACC neurons, reduced pain sensation in our mice model. Overall, our findings support neuromodulation of GABAergic pathways within the nervous system as potential targets for the reduction of inflammatory and neuropathic pain.

Audience Take Away:

- The basic neuroscience of pain perception
- The use of formalin for inflammatory pain model in rodents
- How optogenetics can be utilized for neuromodulation
- Other potential targets for neuromodulation that can be investigated with similar methodology include treatment resistant depression, schizophrenia, vibration/touch sensation and many more possibilities

Biography

Arman John Fijany is a fourth year MD Candidate at the Texas Christian University School of Medicine. He received in BS in Biological Sciences at the University of Southern California.

IN-PERSON

Marina Martinez-Vargas*[1], Miguel A Cruz[2], Edwin Vázquez-Rosa[3], Jing- Fei Dong[4], Andrew Pieper[3], Nobuhide Kobori[5], Promod Dash[5]

[1]Postdoctoral Fellow, Medicine-Thrombosis, Baylor College of Medicine, Houston, Texas, US
[2]Professor and Division Chief, Medicine, Thrombosis Research, Baylor College of Medicine, Houston, Texas, US
[3]Department of Psychiatry, Case Western Reserve University, Cleveland, OH, US
[4]Division of Hematology, Department of Medicine, University of Washington, School of Medicine, Seattle, WA
[5]Department of Neurobiology & Anatomy, University of Texas Health, Texas, US

VWF regulates the expression of adhesion molecules, modulating vascular permeability in endothelial cells after a TBI

Von Willebrand Factor (VWF) is a multimeric protein that mediates thrombo inflammation and has been linked to Traumatic Brain Injury (TBI), specifically in the pathogenesis of early brain injury. Activated endothelium secretes VWF multimers as long hyperadhesive strings. During TBI, the expression of hyperadhesive strings increase significantly. ADAMTS13, a disintegrin and metalloprotease with thrombospondin type-1 repeats, member 13, is a proteolytic factor that reduces the hyperadhesive VWF strings to less adhesive form by cleaving the VWF strings at the A2 domain. In fact, there are experimental studies demonstrating the beneficial effect of intervening with ADAMTS13 after TBI in mice. However, whether the proteolytic fragments of VWF attenuate the severity of TBI remains unknown. Therefore, our research focuses on elucidating the mechanisms by which VWF fragments may regulate the permeability of stimulated endothelium. Fragments of VWF regulate permeability in endothelial cells and play a role in vascular permeability in a TBI mouse model. We employed recombinant proteins encompassing the amino acid sequence of the A1A2A3 domains of VWF. We used biolayer interferometry, immunofluorescence and immunohistochemistry approaches to analyzed vascular permeability during endothelial stimulation. The data shows that Fragments of VWF (A1A2A3 WT and A1A2A3 GOF) modulated permeability, altering the morphology of VE-cadherin, □-actin, and ZO-1. A1A2A3 GOF fragment shown to be effective to rescue permeability at 24 hrs in human microvascular endothelial cells under stimulation with IL-1□. In addition, we noticed a decreased expression of VWF on a mouse brain microvessels after 3 weeks of TBI insult. Based on our experimental data we conclude that VWF is essential to maintain vascular permeability. Our future direction is to evaluate the role of VWF fragments employing a TBI mouse model.

Audience Take Away:

- This presentation will help to understand the mechanisms mediated by VWF in the control of vascular permeability. People who work with TBI can expand their work in order to develop better treatments for people who suffer from this condition. Teachers could develop lines of research for other drugs that can be used to improve the quality of life of these people
- Whether this would help focus research on agents that help improve brain damage caused by TB
- This research is focused on the veteran population but this information can benefit children and young athletes or people who have suffered a severe brain accident
- Today there are not enough treatments to help restore brain damage and this study aims to find new markers with the potential to reduce damage and prevent brain leaks

Biography

Dr. Marina Martinez-Vargas her BS in Microbiology at the University of Puerto Rico (UPR), Humacao Campus. Then, she received her PhD in Biochemistry at UPR, Medical Sciences Campus. She is currently a Postdoctoral Fellow in Neuroscience and Thrombosis at Baylor College of Medicine. She has 6 peer review publications. Her research interest are focus on Traumatic Brain Injury. She is applying for a Career Development Award from the Department of Veteran Affairs. She are proposing to address novel molecular mechanisms associated with the Blood-Brain Barrier (BBB) damage and testing potential reagents capable of improving BBB integrity after TBI.

IN-PERSON

Arman Fijany*[1,2], Nadeem Al-Adli[2], Darryl Narcisse[3], Robert Benkowski[1], Ashutosh Tripathy[3], Samarendra Mohanty[4]

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[2]Texas Christian University School of Medicine, Fort Worth, TX, USA
[3]Opsin Biotherapeutics Bedford, TX, USA
[4]Nanoscope Technologies Arlington, TX, USA

Optogenetic neuromodulation of allodynia and hyperalgesia pain responses in rodent model

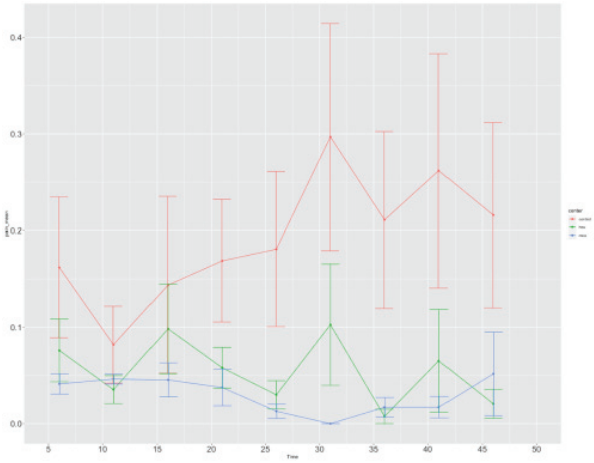
Introduction: While pain is the body's normal physiological response to tissue damage, it is a significant contributor to worldwide disability as well as physical and psychological distress. These deleterious effects are typically dictated by the duration and intensity of pain. Current methods of managing acute and chronic pain largely comprise the utility of controlled opioid medications, which have obvious adverse effects. Chronic pain management may involve alternative techniques such as electronic stimulation and non-narcotic pharmacology such as the use of anticonvulsant drugs for neuropathic pain. However, pain relief from these atypical methods may be variable depending on the patient. Pain is a complex process – involving numerous neurological structures that are integrated into a stepwise fashion from peripheral sensation to central processing. At the level of the spine, the dorsal horn is responsible for the integration of multiple ascending afferent fibers relaying nociceptive pain from their specific dermatomal nerve roots. As such, this location in the pathway of sensation provides a unique opportunity to provide analgesia through alternative methods without the aforementioned deleterious effects associated with current methods.

Methods: Previously, we successfully demonstrated that through the direct stimulation of inhibitory neurons within the anterior cingulate cortex and dorsal root ganglion expressing channelrhodopsin-2, experimental mice could experience significant pain reduction. In this experiment, wild-type mice were used as controls while two experimental groups existed. In one group, a highly sensitive optogenetic actuator multi-characteristic opsin was specifically expressed in inhibitory (GABAergic) neurons via a Glutamic Acid Decarboxylase (GAD) promoter within the epidural space. Another experimental group (HOU) also used a highly sensitive opsin, but this protein was expressed in stimulatory (Glutamatergic) neurons and is expressed via a tryptophan hydroxylase 2 (TPH2) promoter. Using an injection of functionalized Gold Nanorods (fGNRs) with HOU and MCO-encoding DNA, we were able to induce the expression of HOU and MCO in target areas of the nervous system by illuminating them with concentrated bursts of attuned light.

Our novel bluetooth optogenetic pain Modulator device provided optogenetic stimulation with red (630 nm) light at various frequencies and intensities. We used a meshed cage with the Von Frey Assay to measure the pain responses of the Mice. Allodynia was measured via perpendicular monofilament contact, whereas hyperalgesia was modeled by pinprick contact to the hind paw. Reflexive pain responses (Von Frey Assays) were measured over time with a complex pain scoring system that included hind paw retraction, hind paw licking, and spatial preferences.

Results: Optogenetic stimulation of the pain pathways within the epidural space led to a significant reduction in pain in both experimental groups, MCO and HOU groups. In the initial phase, there were significant reductions in pain in both experimental groups not seen in the control groups. Finally, the MCO group experienced more excellent sustenance of pain relief throughout the experiment, while the HOU group demonstrated significant spikes at 30 and 50 minutes. Altogether, these data suggest that in the MCO and HOU groups, optogenetic stimulation within the epidural space provides effective pain relief when compared to controls.

Conclusion: These findings support optogenetic modulation within the epidural space as a potential mechanism of pain reduction.



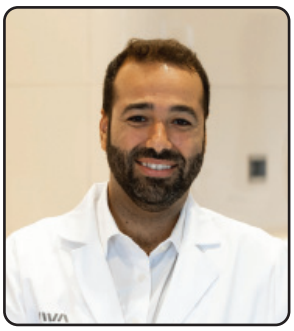
Audience Take Away:

- The pathways involved in pain sensation and their ability to undergo neuromodulation
- Gene delivery of opsin proteins via functional gold nanorods
- The Von Frey assay and its utility for pain responses of allodynia and hyperalgesia in a model for neuropathic pain

Biography

Arman John Fijany is a fourth-year MD Candidate at the Texas Christian University School of Medicine. He received in BS in Biological Sciences at the University of Southern California.

IN-PERSON



Amir Hadanny*[1], Mohammed Elamir*[2]

[1]Aviv Clinics Chief Researcher and Head of Global Clinical Operations, The Villages, Florida, United States
[2]Aviv Clinics Lead Physician, The Villages, Florida, United States

A new outlook on brain injury recovery

In this presentation, Dr. Mohammed Elamir and Dr. Amir Hadanny will present nearly a decade of research in the use of Hyperbaric Oxygen Therapy (HBOT) to treat chronic symptoms related to Traumatic Brain Injury (TBI) and concussion that can be cognitively and physically debilitating. Importantly, this presentation will highlight the gaps in today's treatment protocols for concussions that do not consider Persistent Post-Concussive Syndrome (PPCS).

As both physicians will demonstrate, there is growing evidence related to the regenerative effects of HBOT through induction of the hyperoxic-hypoxic paradox in which repeated intermittent hyperoxia induces many cellular mechanisms that are usually induced during hypoxia, including HIF-1-alpha and VEGF. This leads to stem cell production and improvement in tissue oxygenation, producing improved mitochondrial metabolism, and anti-apoptotic and anti-inflammatory effects. This method ultimately makes HBOT promising for addressing brain injury in adults and children by inducing neuroplasticity, angiogenesis, and neurogenesis. This method has been evaluated for nearly a decade through various means of prospective, randomized, crossover-controlled clinical trials in adults, adolescents, and children and published in leading, national, peer-reviewed medical journals.

Here is a brief synopsis of the research and results to be presented and discussed.

A randomized, sham-controlled, double-blind 2022 trial included children ages 8-15 with PPCS who received 60 daily HBOT treatments. Participants were analyzed pre-and post-treatment with cognitive and neuropsychological tests, health and behavior inventories, quality of life assessments, and DTI MRI imaging.

Results: Children in the 2022 treatment group had a significant increase in cognitive function including general cognitive score ($d=0.598$, $p=0.01$), memory ($d=0.480$, $p=0.02$), and executive function ($d=0.739$, $p=0.003$), as well as improved behavioral symptoms. Outcomes were correlated with significant microstructural improvements in the insula, supramarginal, lingual, inferior frontal, and fusiform gyri.

A 2018 retrospective analysis included 154 patients suffering from chronic neurocognitive damage from TBIs. All patients within the study were treated with 40-70 daily hyperbaric sessions, five days a week. Researchers administered computerized pre- and post-HBOT cognitive evaluations and SPECT scans.

Results: significant improvement in all cognitive domains, particularly in attention and memory, with mean changes of 8.1 ± 16.9 ($p<0.00001$) and 6.8 ± 16.5 ($p<0.0001$). SPECT images illustrated significant improvement in the postcentral cortex, prefrontal areas, and temporal areas.

Another retrospective analysis in 2015 reviewed perfusion MRI scans and clinical cognitive scores of 10 adult post-concussion syndrome patients taken before and after an HBOT protocol of 60 daily sessions, five days per week. Patients had sustained their injuries with a mean of 10.3 ± 3.2 years prior to treatment.

Results: brain perfusion analysis showed significantly increased cerebral blood flow and volume, suggesting that HBOT induces cerebral angiogenesis to promote brain injury healing even years after the initial injury. Subjects also experienced significant improvement in global cognitive scores ($p = 0.007$).

Conclusion: The overarching conclusion of these studies demonstrates that the hyperoxic-hypoxic HBOT protocol was associated with overall cognitive improvement and brain health recovery.

Audience Take Away:

- Primary learning: Attendees will be presented with an in-depth look and analysis of the regenerative and neuro-rehabilitative effects of HBOT specific to persistent post-concussive syndrome and traumatic brain injury.
- Secondarily: Attendees will learn that different levels of controlled oxygen can improve future cognitive decline related to other brain-damaging diseases such as stroke, dementia, and Alzheimer's Disease
- Lastly, attendees will learn how innovative HBOT treatment programs are applied (and backed by clinical research) as part of a comprehensive brain health and wellness approach to treat other debilitating neurological conditions and diseases such as PTSD, long COVID, and mild cognitive impairment.

Biography

Dr. Amir Hadanny is a board-certified neurosurgeon, researcher and physician at Aviv Clinics, and Chief Medical Research Officer at the Sagol Center for Hyperbaric Medicine and Research. For the past decade, he has worked alongside Dr. Efrati researching neurorehabilitation, neuroplasticity, and physiology, publishing more than 25 papers on the effects of HBOT on cognitive and physical performance. Before joining the Sagol Center, Dr. Hadanny was Chief Resident in the Galil Medical Center neurosurgery department. He earned his MD from Tel Aviv University and his Ph.D. in Bioinformatics and Machine Learning from Bar Ilan University.

Dr. Mohammed Elamir is an Aviv Clinics physician with over ten years of experience in Internal Medicine. Prior to joining Aviv, he spent five years practicing Internal Medicine at the MM Jersey City Breathing Center while owning his medical spa in New Jersey. He graduated from Rutgers University and attended St. Matthew's University School of Medicine. He completed his Internal Medicine Internship and Residency at RWJ-Barnabas: Jersey City Medical Center Program where he was Chief Resident. He is a Fellow of The American College of Physicians, The American Board of Internal Medicine, and The American Board of Aesthetic Medicine.

IN-PERSON

Ulrich Sprick*[1], Martin Kohne[2]
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MRI- navigated Transcranial Pulse Stimulation (TPS) as an alternative option for severe treatment-resistant depression– a case report

Patients with severe therapy-resistant depressive disorders repeatedly pose enormous treatment challenges in psychiatry and neurology. Transcranial Pulse Stimulation (TPS) as a new non-invasive therapy method could represent a new alternative to standard treatment such as ECT, magnetic stimulation or deep brain stimulation. In contrast to ultrasound stimulation (tFUS) TPS uses shock waves. These shock waves allow an application to superficial brain structures as well as into deep brain areas without the induction of any thermal side effects. The stimulation of the target areas is MRI- guided with a similar precision as in a stereotactic procedure.

TPS is currently approved for the treatment of Alzheimer's Disease. Since AD patients repeatedly reported significant improvements of their mood, a healing attempt was carried out in a patient with a severe therapy-resistant depression. An 81-year-old inpatient suffered from a major depressive disorder (lasting for longer than 18 months). All pharmacological approaches with different antidepressants (including ketamine) also in combination treatment and adjuvant therapy did not improve the status of the patient. ECT also did not lead to any positive change. The TPS-treatment was performed as a healing attempt. The patient received bilateral stimulation of the nucleus accumbens (the “reward center” of the brain) and the frontodorsal cortex (bilaterally 3,000 pulses per session, 0.2 mJ/mm2 per pulse, frequency 4 Hz). The navigated stimulation was carried out over a period of 2 weeks (with 3 sessions per week) by a neurolith apparatus (Storz Medical, Switzerland). In addition to the clinical assessment of depressive symptoms, standard tests with a TFDD and CERAD were carried out. The patient was examined and tested before TPS stimulation as well as 2 and 6 weeks later. A dementia syndrome was not present in the patient.

TPS stimulation led to an exceptional significant improvement in mood, which was also reflected in the TFDD (18/20 to 6/20). This improvement persisted for over 6 weeks. Side effects were not reported by the patient. This case shows that MRI-navigated TPS can be considered as an alternative method for therapy-resistant major depression. To determine the exact mechanisms of action of TPS further investigation is needed. In a rodent model it could be shown that TPS induced an increase of Nitric Oxide (NO)- levels. Both, the release of trophic factors and a temporarily opened Blood-Brain-Barrier (BBB), seem to play a critical role. Thus a transient opening of the BBB might help to potentiate effects of administered pharmaceuticals contributing to an improvement of brain function.

In summary TPS is a very promising option as an adjunctive therapy to a state-of-the-art treatment which may achieve a reduction of depressive symptoms as well as an improvement of cognitive functions and quality of life.

Audience Take Away:

- Presentation of a new effective method of non-invasive brain stimulation with very low side effects
- Beside the case report of a patient with a severe depression data of own treatments of Alzheimer's disease with TPS will be included
- Discussion of the working mechanism of TPS

Biography

Prof. Dr. med. Dr. rer. nat. Dipl.-Psych. Ulrich Sprick studied Medicine and Psychology at the University of Dusseldorf. He is an associated professor at the Medical Faculty of the University of Dusseldorf. Trained as a specialized psychiatrist and an expert psychologist in neuropsychology he works as Deputy Medical Director and Head of the Department of Dayclinics and Out-Patients of the Alexius/Josef Clinic in Neuss (Germany). He has been working in brain research for more than 20 years with special interests in brain stimulation, neuoplasticity, trophic factors, endogenous opiates and memory. From the clinical perspective his actual research interests include treatment of Alzheimer's Disease and depression, nonvisual effects of light and telemedicine.

Aygun BadaLova
University College London, United Kingdom

Could we see alzheimer's disease through the eye?

One of the most common and prevalent neurodegenerative disorders are Alzheimer's and Dementia which are considered significant causes of mortality and morbidity in today's society particularly among the elderly population(Erkkinen, Kim et al. 2018). The etiologic and pathophysiologic factors of neuro-degenerative disorders are still complicated and unrecognized. In last decade the overall mortality rate for deaths cause by Dementia and Alzheimer's Disease has been generally increasing year-on-year. One of concerning statistics reported by office for national statistics which indicates approximately 52% of increase in amount of people who diagnosed with dementia. Indeed it is very threatening statistics in deaths of any health condition. From statistical point of view such huge increase on these disorders make and urgent need to understand the mechanisms behind the onset and progression of these heterogeneous diseases (Fig 1). Currently diagnosing Alzheimer's disease is also challenging due to lack of biomarkers. One of the pathological factors of Alzheimer's disease include the accumulation of proteins such as hyper phosphorylated tau and amyloid protein in hippocampal areas of the brain. The detection of these pathological biomarkers are conducted either by performing cerebrospinal fluid analysis, brain imaging or post-mortem brain tissues under microscope. However, these methods are not easily accessible and largely available due to different reasons. These include challenges in collecting certain samples, lack of post-mortem tissues and high-cost of experiment.(Guidoboni, Sacco et al. 2020).

Many researches and investigations on neurodegenerative disorders suggest that when neurodegeneration process starts in the brain, this alteration can also influence the eyes. In fact human eye is directly linked to the brain via the optic nerve. The brain is the main area of our eyes and our visual system which mainly consist of optic nerve and HYPERLINK "https://www.aao.org/eye-health/anatomy/retina-103" retina and these two main part of eyes one of the brain tissues. Alternatively when there is a damage on different brain cells, these atrophic changes also affect to the function of retina. Human eye illustrates a very spectacular window to the brain which can be easily examined by ocular imaging. Researches also indicate that individuals suffering from neurological disorders and several other neurological diseases often exhibit significant structural and vascular changes in the visual field.(T Reed, Behar-Cohen et al. 2017) In fact vascular changes in the eye is also associated vascular changes in the brain (Heringa, Bouvy et al. 2013) This association is not surprising because the eye as an organ is a protrusion of the brain and the origin of our visual system starts from brain .In addition the ophthalmic artery plays one of the main role in brain as well as providing eye with blood. Retina also responsible for various characteristics with the cerebral microvasculature and the neurons of the central nervous system.

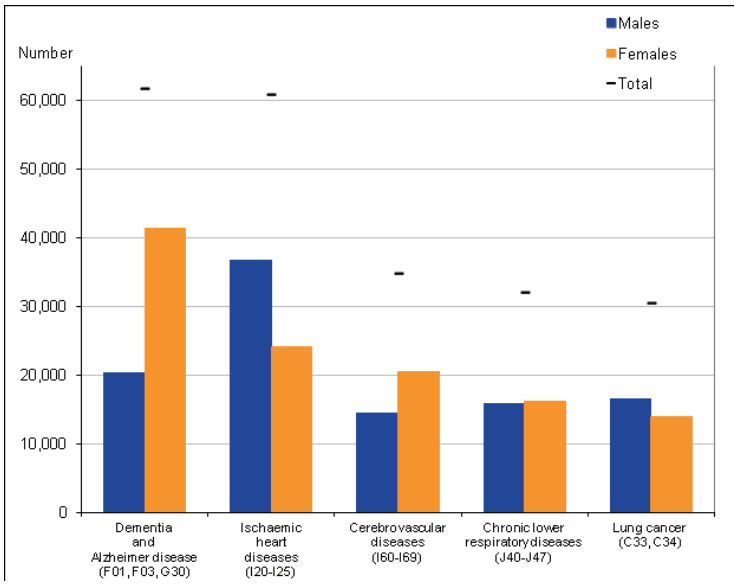

Figure 1: The cause of death groups used here are based on a list developed by the WHO, modified for use in England and Wales. Source: Office for National Statistics

According to various research many patients diagnosed with Alzheimer's report impairments with visual symptoms. (Fiorio, Tinazzi et al. 2006)These facts indeed had been an increased interest in finding the specific peculiar optholmalogic biomarkers. In fact, the contribution of ocular measurements is very wide in terms of early diagnosing many morbid diseases including heart,stroke.The usage of eye measurements have been considered as a significant tool of biomarkers for the prediction of neurodegenerative disorders. Another reason is that the eye is the only place in the human body where structural and functional vascular features can be observed and measured easily and non-invasively down to the capillary level (Harris et al., 2003; Weinreb and Harris, 2009).

Last decade the relationship between neurodegenerative disorders and eye had been n main interest area of scientists. Recent studies have revealed that there are many similarities linking glaucoma and AD. Studies suggest that Alzheimer's Disease and glaucoma should be considered age-related neurodegenerative diseases, bearing in mind the common features of both diseases, including risk factors and pathophysiological mechanisms.(Mancino, Martucci et al. 2018) Furthermore, recent studies suggest that at the molecular genetic level, AD and age-related macular degeneration (AMD) share common, pathological signaling defects and disease mechanisms.(Biscetti, Luchetti et al. 2017) There is increasing evidence that □-amyloid, the main component of senile plaques that characterize AD, is also a vital component and a hallmark of AMD.(Frost, Guymer et al. 2016)

One thing is that AMD is a retinal disease and affect mostly retina , research suggest that AD also damages brain cells as well as the retina. Both of these age-related diseases initially affect different parts of the central nervous system, but are generally similar in terms of abnormal extracellular deposits, metabolic and oxidative stress, neuroinflammation, and microvascular abnormalities.(Sant'Anna, Navarro et al. 2016)

Another reason is that both diseases occur almost after a certain age and are mostly found in the elderly population, which further strengthened this similarity.(Giorgio, Zhang et al. 2018) in addition the leading cause of irreversible blindness (glaucoma) is progressive degeneration of the optic nerve and corresponding death of retinal ganglion cell.

There is strong evidence to suggest that the proliferation of neurodegenerative diseases and their specific ocular biomarkers play a crucial role in the development of retinal dysfunction or loss of vision function. (Reitz and Mayeux 2014)

VIRTUAL

Dell G Mars
Nursing, Southeastern Louisiana University School of Nursing, Baton Rouge, Louisiana, USA

Lived experience of the African American informal caregivers of a family member with Alzheimer's Disease and related Dementia

Purpose: The purpose of this qualitative study was to describe the lived experience of African American informal caregivers of family members of Alzheimer's Disease and Related Dementias (ADRD) in a home environment.

Design and Method: This phenomenological descriptive qualitative study guided by Husserl's philosophy of the phenomenology was used to interview 16 African American informal caregivers in southeastern United States. Colaizzi's method of data analysis was used to analyze the data. The participants were solicited by word of mouth from a local church, an adult day care center, a physician's clinic, a home community-based service, and a respite care program. The participants were interviewed using a semi- structured questionnaire; each interview lasted an average of 60 minutes.

Findings: Four thematic areas reflecting caregiver understands of caregiving were identified: a) sense of obligation, b) arduous journey, c) sentinel events, and d faith in God. Findings indicated that caregivers needed to be well-informed concerning the demands of caregiving and needed more assistance with the task of delivering care. Informal caregivers lacked support, knowledge, and guidance.

Conclusion: This study offers a fresh, in-depth insight into how African American informal caregivers understood their experiences with caregiving. While caregiving for a family member with a debilitating disease like Alzheimer's and related Dementias can be challenging and can cause physical, emotional, and potential financial strain, it is not and does not have to be the complete experience for caregivers who willingly undertake the role. Implications for the discipline of nursing include emphasis on family assessment, teaching, awareness of resources, and collaboration with health care teams. Family assessment of the caregiving situation can lead to positive outcomes for the caregiver and care recipient.

Audience Take Away:

- The audience will be able to use that they learn because they will be made aware of a broad range of the needs of the informal African American caregivers of a family member with ADRD. The findings of this study can provide valuable information to improve services for African American informal caregivers. Nurses need to be aware of family needs and accurately assess them so that they can be addressed
- This will help the audience be more proficient in their jobs because nurses should be aware that the needs of the African American caregivers with a family member with ADRD are complex. It is the role of the nurse to assess the needs of the caregiver and collaborate with physicians and other members of multidisciplinary teams to provide intervention for the needed support of the caregiver
- I believe this research could be used to expand other faculty research or teaching by further refining the nurses' understanding of caregiver challenges. Studies on the impact of full-time employment, being part of the sandwich generation, and the lack of socialization on the well-being of informal caregivers should be conducted. Conduct studies for caregivers outside of the community resources networks

Biography

Dr. Dell Mars received an MSN in Nursing from Southern University and A&M College in 1999 and received her PhD in Nursing degree in 2015 from Hampton University. She is employed as an assistant professor at Southeastern Louisiana University School of Nursing. Dr. Dell Mars served as a keynote speaker at several Global Episteme Conferences in Nursing. She is employed as an assistant professor of nursing at Southeastern Louisiana University School of Nursing. Dr. Mars is a published author and actively disseminate nursing research at numerous professional local, national, and global nursing conferences.

IN-PERSON

Pavel Novak
Business Development, Storz Medical AG, Tägerwilen, Switzerland

Alzheimer's disease treatment with TPS (Transcranial Pulse Stimulation) significantly improves patient's cognitive functions

Low intensity shockwaves proved to be efficient for the treatment of non-unions, tendon and muscular pain, wound healing, heart insufficiency, erectile dysfunction, aesthetic since 1990. The working principle is the mechanical stimulation of biological processes called mechanotransduction. Transcranial Pulse Stimulation (TPS) uses shockwave pulses for mechanical stimulation of the brain tissue.

Alzheimer's disease patients have been treated with TPS since 2010. The treatment consists of a booster block of 6 treatment sessions within 2 weeks and one re-treatment session per month after the initial 3 months. 6000 shockwave pulses with energy flux density of 0.2mJ/mm2 are applied in one session. There is no need to shave patient's hairs.

The CERAD score is improved by 10-17% within the first 3 months. The long-term data exists for more than 3 years. The results to date show that the monthly re-treatment is sufficient for maintaining the improvement for at least one year. Thereafter, dependent on patient's disease progress it might be necessary to repeat the initial booster block of 6 sessions in 2 weeks. Meanwhile there are over 100 clinical sites using this method (Neurolith, Storz Medical AG) resulting in over 1500 treated patients with more than 25'000 treatment sessions. The treatment is painless and very well accepted, with satisfaction rate of up to 80%. It is safe and effective. There have been no relevant negative side effect observed.

The regulatory clearance in Europe (CE mark) is based on two center clinical trial with 35 patients. In spite of the numerous successful clinical applications, further scientific data are needed. There are placebo controlled clinical trials ongoing. Further indication are under evaluation.

Audience Take Away:

- The presented treatment with TPS (Neurolith) is cleared for sale in the EU and many other countries
- The procedure will be described. It can be simple performed by skilled medical professionals
- The TPS treatment is meanwhile performed at more than 100 clinical sites very successfully
- Further clinical trials for broader database are required
- Further indication are under evaluation

Biography

1974 and 1980: Dipl.Ing. and Dr.-Ing. in Electrical Engineering, Munich Technical University, Germany Followed by Postdoc in Biomedical Engineering
1974–1985: Fraunhofer Institute for Solid State Technology, Munich, Applied research and development
1985–1990: Dornier Medizintechnik GmbH, Germering, Head of Electronics Development.
1990–2003: Storz Endoskop Produktions GmbH, Schaffhausen, Switzerland, Head of Development and Production Medical Electronics and Technical Endoscopy.
2003-2016: Storz Medical AG, Tägerwilen, Switzerland, Head of Product Development.
2016-2018: Head of Product Management
Since 2019: Science & Technology Director
Approx. 90 patents and more than 100 scientific papers and presentations.
Member of BMT/VDE, ISMST, ESSM, ASLMS, ISPRM, SfN, ISAART and IEEE.

IN-PERSON

Xiaodong Cheng*[1], Ni Ye1, Jennifer Cruz[1,2], Xiaoyan Peng[1], Jinyun Ma[1], Aiming Zhang[3]

[1]Institute of Clinical Immunology, Yue-Yang Hospital of Integrative Medicine, Shanghai University of Traditional Chinese Medicine, Shanghai, PR China
[2]Doctoral Program of Acupuncture & Oriental Medicine, The Atlantic Institute of Oriental Medicine, USA
[3]Department of Neurology, Min-Hang Hospital of Integrative Medicine, Shanghai, PR China

Remyelination is enhanced by astragalus polysaccharides through inducing the differentiation of oligodendrocytes from neural stem cells, in cuprizone model of demyelination

Demyelination is the hallmark of Multiple Sclerosis (MS). Promoting remyelination is an important strategy to treat MS. Our previous study showed that Astragalus Polysaccharides (APS), the main bioactive component of astragalus membranaceus, could prevent demyelination in experimental autoimmune encephalomyelitis mice. To investigate the effects of APS on remyelination and the underlying mechanisms, in this study we set up a cuprizone-induced demyelination model in mice and treated them with APS. It was found that APS relieved the neurobehavioral dysfunctions caused by demyelination, and efficaciously facilitated remyelination in vivo. In order to determine whether the mechanism of enhancing remyelination was associated with the differentiation of Neural Stem Cells (NSCs), biomarkers of NSCs, astrocytes, oligodendrocytes and neurons were measured in the corpus callosum tissues of mice through real-time PCR, western blot and immunohistochemistry assays. Data revealed that APS suppressed the stemness of NSCs, reduced the differentiation of NSCs into astrocytes, and promoted the differentiation into oligodendrocytes and neurons. This phenomenon was confirmed in the differentiation model of C17.2 NSCs cultured in vitro. Since sonic hedgehog signaling pathway has been proven to be crucial to the differentiation of NSCs into oligodendrocytes, we examined expression levels of the key molecules in this pathway in vivo and in vitro, and eventually found APS activated this signaling pathway. Together, our results demonstrated that APS probably activated sonic hedgehog signaling pathway first, then induced NSCs to differentiate into oligodendrocytes and promoted remyelination, which suggested that APS might be a potential candidate in treating MS.

Biography

Dr. Xiaodong Cheng (M.D., Ph.D.) is currently a full Professor of Immunology, Neurology, and Integrative Medicine; Director of Institute of Clinical Immunology, Yue-Yang Hospital of Integrative Medicine, Shanghai University of Traditional Chinese Medicine (TCM). He is also an Adjunct Professor of The Atlantic Institute of Oriental Medicine, USA. He is not only a basic research scientist but also a clinical physician of TCM as well. Dr. Cheng got his M.D. and Ph.D. degree at Fudan University in 1996, and had his postdoctoral training at Shanghai Institute of Cell Biology, Chinese Academy of Sciences. Since 2000, he was awarded the Max-Planck Society Scholarship and was working as a visiting scientist at Max-Planck Institute of Neurobiology, Germany. After that, he moved to the USA to pursue his research in Thomas Jefferson University, Pennsylvania; Blood Research Institute, Wisconsin; and Rutgers University, New Jersey; respectively until 2009. From 2009 to 2015, Dr. Cheng was appointed as a Chair Professor and Director of National TCM Great Master Training Program; Director of Department of Cellular and Molecular Immunology, School of Life Sciences and Technology; Director of Research Center of Pharmacology & Toxicology for TCM, Li-shui Institute of Traditional Chinese Medicine; Tongji University right now, Dr. Cheng is an active member of the American Association of Immunologists (AAI), USA; active member of The European GP-TCM Association (GP-TCM); the secretary-general of Shanghai region of The Consortium for Globalization of Chinese Medicine (CGCM); and active member of World Federal of Chinese Medicine Society (WFCMS). Dr. Cheng's research interests are now focused on: 1, Clinical trial and basic study on therapeutic approaches with traditional Chinese medicine in treating autoimmune, infectious, and cancer diseases. 2, Targeting therapy to cancer diseases by Chinese medical herbs in combination with monoclonal antibody and its related immunological mechanisms; 3, Immunopathogenesis of CNS autoimmune inflammatory diseases; 4, Globalization of traditional Chinese medicine and its cross-talking with western medicine.

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24-26 OCT

DAY 01

POSTERS

6TH EDITION OF

INTERNATIONAL CONFERENCE ON

NEUROLOGY AND BRAIN DISORDERS

IN-PERSON

Alsu Zagorulko*[1], Alan R Hirsch[2], Pavel Sinyagovskiy[3]
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[2]Illinois Center for Neurological and Behavioral Medicine, Des Plaines, IL; Smell & Taste Treatment and Research Foundation, Chicago, IL
[3]Yuma Regional Medical Center, Yuma, AZ

Mortality, length of stay, and cost of hospitalization among adult patients with multiple sclerosis: Results from the national inpatient sample

Introduction: The study was performed to evaluate factors associated with in-hospital mortality, Length Of Stay (LOS), and hospitalization cost among adults with Multiple Sclerosis (MS).

Methods: We used the National Inpatient Sample (2019) to identify adult hospitalizations with MS. Logistic, Poisson, and linear regression models were constructed to evaluate mortality, LOS and log transformed cost of hospitalization respectively. Dynamite plot was added to determine the most common primary diagnoses among the entire cohort and non-survivors.

Results: There were 151,635 hospitalizations with MS nationwide, and the inpatient mortality rate was 1,58%. Non-survivors were predominantly older (median age 66.0 years, IQR (Interquartile Range) 57.75-74.00), female (68%) and Caucasian (81.6%).

Using clinical classification software diagnostic categories, bacterial infections (12%) and septicemia (11%) were the most common primary diagnostic groups among the entire cohort and among those who died (28% each). Respiratory failure (adjusted Odds Ratio (aOR) = 10.8, 95%CI (confidence interval): 8.4-13.9, p=0.001) was strongly associated with in-hospital mortality. Diabetes Mellitus (aOR = 0.6, 95%CI: 0.4-0.8, p=0.001) was associated with decreased in-hospital mortality, although univariate model failed to find such association (OR = 0.95, 95% CI:0.76-1.18, p=0.62).

Median length of stay was 4 days (IQR 2-6), while the median cost of hospitalization was $36,844 (IQR $20,871-$66,480). Patients with Asian race had the highest cost of hospitalization (median $53,005, IQR $27,749-$93,653, p=0.001). Multivariable linear regression with log transformed hospitalization charge re-affirmed that Asian race was associated with higher hospitalization cost (adjusted regression coefficient (β) = 0.192, 95%CI: 0.087-0.297, p=0.001), as well as private insurance (adjusted β=0.041, 95%CI: 0.017-0.165, p=0.001). Patient-level factors associated with higher hospitalization costs were stroke and transient ischemic attack (adjusted β=0.224, 95%CI: 0.172-0.276, p=0.001), and respiratory failure (adjusted β=0.273, 95%CI: 0.242-0.304, p=0.001).

In a restricted cohort, multivariable Poisson regression model showed that Black race was associated with longer LOS (adjusted incidence rate ratio (aIRR)=1.043, 95%CI: 1.02-1.067, p =0.001), while private insurance was associated with shorter LOS (aIRR=0.952, 95%CI: 0.932-0.972, p=0.001). Clinical factors associated with longer LOS were pressure ulcer (aIRR=1.229, 95%CI: 1.201-1.257, p=0.001) and respiratory failure (aIRR=1.224, 95%CI: 1.195-1.254, p=0.001).

Conclusion: Bacterial infections and septicemia were the most common primary diagnoses among adults hospitalized with multiple sclerosis. Bacterial infections, followed by septicemia were top two primary diagnoses for non-survivors. Respiratory failure was strongly associated with in hospital mortality.

Audience Take Away:

- Bacterial infections are the most common diagnoses among adults admitted to the hospital with a history of multiple sclerosis
- Respiratory failure was strongly associated with in-hospital mortality
- It may be possible that intervention targeting early recognition or optimal management of infections may lead to improved outcomes
- How will this help the audience in their job? Is this research that other faculty could use to expand their research or teaching? Does this provide a practical solution to a problem that could simplify or make a designer's job more efficient? Will it improve the accuracy of a design, or provide new information to assist in a design problem? List all other benefits
- Study lists contemporary epidemiology and patient outcomes of patients with multiple sclerosis who have been admitted to the US hospitals
- The study is observational and should be served as hypothesis generating. Our work doesn't provide a "practical solution" to the problem of reduction of mortality, Length Of Stay (LOS), or hospitalization costs. The primary aim of the study was to describe the relationship between pre-selected outcome variables such as mortality, LOS, and charges and a group of dependent variables

Biography

Dr. Alsu Zagorulko studied Medicine at Russian National Research Medical University, named after N.I. Pirogov, Moscow, Russia, and graduated as M.D. in 2017. She then began preparation for USMLE Steps to be able to practice Medicine in the United States. She moved there in 2019, passed USMLE Steps, and acquired an ECFMG certificate in 2021. She is interested in Psychiatry and currently rotating in mental health hospitals and clinics around the States. At the same time, she's doing research in Neurology and Psychiatry. She is applying for the Psychiatry Residency this year.

IN-PERSON

Solomon Nittala*[1], Jeremy Eckes[2], Sadek Debwan[2], Karla Romero[3], Saloni Shirke[4], Sana Hussaini[4], Kester Nedd[5]

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The apt diagnosis of progressive supranuclear palsy with frontal predominance: A case report

Originally described in 1964, Progressive Supranuclear Palsy (PSP) is a neurodegenerative tauopathy with a prevalence of approximately 45% among other parkinsonian-plus syndromes. PSP can be categorized into various phenotypes among which 6% belong to the PSP-frontal predominant (PSP-variant [1,2]. PSP-F presents primarily with vertical supranuclear palsy and frontal-cortical symptoms including apathy, bradyphrenia, impulsivity, dysexecutive syndrome, and reduced phonemic verbal fluency [3].

Here we describe the case of a 75-year-old Caucasian female with a past medical history of Parkinson's Disorder (PD), Anxiety, and Depressive disorder who presented for altered mental status after a syncopal episode. The patient had experienced multiple unprovoked falls in the past three years with PD symptoms (tremors and gait abnormalities) that were resistant to levodopa. She reported difficulty looking upwards, resulting in falls when reaching for overhead objects. As per family, she also experienced multiple crying spells during inappropriate times. Neurological exam was significant for an asymmetric resting tremor of the right hand, a rolling tremor of the neck, and limited passive range of motion of the bilateral lower extremities with 3/5 motor strength in all four extremities. Cranial Nerve (CN) testing for CN III, IV, VI showed vertical gaze palsy with intact extraocular muscles, normal convergence, and no nystagmus. Neurological examination of language revealed no deficits in either receptive or expressive aphasia and naming repetition during patient interviews; however, the patient was noted to have a waxing and waning course of expressive aphasia during the hospital course by other staff. The rest of the physical examination was normal. Computed tomography of the brain showed atrophy of the cerebellum, fronto-temporal cerebral regions, and the brainstem (with a "hummingbird sign") without evidence of acute processes. A probable diagnosis of parkinsonism secondary to PSP was made based on the National Institute of Neurological Disorder and Stroke, and the Movement Disorder Society criteria for PSP. We believe our patient had the PSP-F variant due to a history of pseudobulbar affect, expressive aphasia, and imaging findings. A conservative approach was used to manage the patient with a focus on reducing polypharmacy and establishing fall precautions at home.

The diagnosis of PSP should be made promptly but can be challenging in early phases due to confounding factors, incomplete history, and limited accuracy of diagnostic tests. In PSP-F, magnetic resonance imaging can confirm brain atrophy (specifically of the midbrain and superior cerebellar peduncles) and demonstrates a "hummingbird or penguin body" on the brainstem. It can also help exclude other differentials [4]. Positron emission tomography or single-photon emission computerized tomography may exhibit hypometabolism of the frontal cortex, caudate head, thalami, cingulate gyri, and midbrain [5]. While imaging findings are useful in identifying disease course, they are non-specific and only autopsy can definitively confirm the diagnosis [3]. Recognizing the signs and symptoms early in the disease is essential to avoid misuse of antidepressants, neuroleptics, anticholinesterase inhibi tors, and dopaminergic agents which have not shown any significant benefits [6].

Audience Take Away:

- Signs and symptoms of Progressive Supranuclear Palsy (PSP) with Frontal Predominance among Parkinson Plus Syndromes
- To better equip clinicians with the necessary tools to identify, diagnose, and manage PSP
- Review of radiographic studies in diagnostic evaluation of Parkinson Plus Syndromes

Biography

Dr. Nittala began studying neuroscience as a high school student at the Illinois Mathematics and Science Academy where his first research experience was at Northwestern University with fMRI of Chronic Phantom Limb Pain. He studied at the University of Illinois at Chicago, double major in Psychology and Biology. He received Master's degree from the University of Texas at Dallas in Applied Cognition and Neuroscience with a thesis on emotion recognition using Event-Related Potentials on Electroencephalogram (EEG). Dr. Nittala completed medical school at Nova Southeastern University and is now a Neurology Resident Physician at Larkin Community Hospital in Miami, Florida.

IN-PERSON

Elita Delbruck*[1,2], Ai Ohno[1,2], Natasha Anand, Saloni Gupta[1], Nicolas Thor, Farbod Farmand[1]

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Rapidly progressive sporadic Creutzfeldt-Jakob Disease (CJD): A case report and literature review

Prion diseases are rare and rapidly progressive neuro-degenerative disorders that are poorly understood. Universally fatal, diagnosis primarily focuses on the excluding other treatable diseases. This case of probable sporadic Creutzfeldt-Jakob Disease (sCJD) in an otherwise healthy 61-year-old male outlines presenting symptoms and disease course. Diagnosis was supported by a positive RTQuIC and detection of 14-3-3 and T-tau proteins in the cerebrospinal fluid. Notable lab findings included oligoclonal bands and elevated natural killer cells. Treatment with corticosteroids, IVIG, levetiracetam, and carbidopa-levodopa were ineffective, while benzodiazepines provided symptomatic relief. This case report and literature review identifies the importance of including sCJD as a differential for patients presenting with rapidly progressive motor and cerebellar dysfunction with constitutional symptoms for early identification of the disease.

Audience Take Away:

- Recognition of the atypical characteristics of an especially rapid clinical course of a patient with CJD for early accurate diagnosis
- Diagnostic value of various tools used in aiding diagnosis in this patient such as protein 14-3-3, t-tau protein, RT-QuIC, EEG, and MRI
- Novel findings of diagnostic testing in this patient's cerebrospinal fluid that can potentially aid in understanding the pathophysiology and enhancing diagnostic tools for CJD
- Variable efficacy of treatment with levodopa-carbidopa, levetiracetam, corticosteroids, IVIG, and clonazepam in this patient

Biography

Elita Delbruck studied Cognitive Neuroscience at the University of California, Irvine where she joined the Visual Perception and Neuroimaging Lab in the School of Social Sciences and later also joined the Vawter Lab in the Department of Psychiatry and Human Behavior. She graduated with a Bachelor of Science and several honors awards in 2017. She began medical school at the California University of Science and Medicine in 2019, where she is currently in her last year of pursuing her MD and a residency training program in neurology. She has multiple research publications in neuroscience and aspires to continue her career in clinical neurology research.

IN-PERSON

Luis A. Sierra*, Karen Hildebrand, Clementina J. Ullman, BA, Julia S. Dierker, Samuel A. Frank, Simon Laganiere
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A mild cognitive impairment diagnostic tool in comparison to the unified huntington's disease rating scale: Cognitive battery

Background: A diagnosis of manifest Huntington's Disease (HD) is based primarily on motor symptoms, but early cognitive decline is common in the pre-manifest HD period (preHD). Early symptom detection in HD has important implications for monitoring both disease progression and the effects of disease-modifying treatment trials. Identifying the assessments with the highest sensitivity for capturing early cognitive changes in preHD, therefore, remains a priority.

Objectives: The Loewenstein Acevedo Scales of Semantic Interference and Learning (LASSI-L) is a novel test that captures simultaneous deficits in executive functioning, processing speed, and memory retrieval. Our goals were to determine whether the LASSI-L could detect cognitive deficits in preHD and compare this to the Unified Huntington's Disease Rating Scale (UHDRS): Cognitive Battery.

Methods: We administered the LASSI-L to 14 preHD participants and 12 healthy controls, matched for age, sex, and education, recruited from the Huntington's Disease Society of America Center of Excellence at Beth Israel Deaconess Medical Center (BIDMC). As a comparison, we administered the UHDRS cognitive battery which includes the Stroop, Symbol Digit Modalities Test (SDMT), and Category Fluency (Animals).

Results: 6 of 7 sections on the LASSI-L captured significant group differences: proactive semantic interference (PSI) ($p < 0.001$), failure to recover from PSI (frPSI) ($p = 0.035$), retroactive semantic interference (RSI) ($p = 0.020$), delayed recall ($p < 0.001$), B1 cued recall intrusions ($p = 0.007$), and B2 cued recall intrusions ($p = 0.033$). Using (FDR) < 0.05, PSI, RSI, delayed recall, and B1 cued recall intrusions remained significant. In comparison, from the UHDRS cognitive battery only the three sections of the Stoop were significant. Stroop Word Reading ($p < 0.001$), Stroop Color Naming ($p = 0.005$), and Stroop Interference ($p = 0.041$). Using (FDR) < 0.05, only Word Reading and Color Naming remain significant.

Conclusions: The LASSI-L time-efficient and sensitive task captures early cognitive decline in pre-manifest HD and outperforms several commonly used neuropsychological tests. These results show that the LASSI-L would be a useful addition to current protocols in preHD-related studies.

Audience Take Away:

- The audience will learn that although Huntington's disease is primarily thought of as a motor manifest disease, cognitive decline precedes these symptoms and should be focused on as an early biomarker for manifestation
- Although the UHDRS cognitive battery has been the standard since 1996, it would be important to update the battery with paradigms that significantly capture deficits not only initially, but over time
- Proactive Semantic Interference seems to be a predominant marker in deficits of cognition with pre-manifest HD
- Lastly, the LASSI-L is a cost effective, time-efficient, and sensitive assessment that would be practical to use in a clinic setting

Biography

Luis A. Sierra studied clinical psychology at Barry University, Miami Shores, and graduated with MS in 2020. While obtaining his MS, he worked as a psychometrist at the University of Miami's Center for Cognitive Neuroscience and Aging under Dr. David Loewenstein. He then joined the research group of Dr. Samuel Frank and Dr. Daniel Press at BIDMC's Program to Advance Innovative Neurodegenerative Therapies as a Clinical Trials Specialist for Huntington's, Parkinson's, and Alzheimer's disease. His next career goal is to obtain a Ph.D. in Neuropsychology.

IN-PERSON

Brittany Liberati[2], John Pamula[2], Sarah Branch[1,2], Ananya Chandra, BS[1,2], Rebecca Lees * [1,2], Leonard Javick[1,2]

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Improving stroke quality metrics at Robert Packer hospital

Stroke is the number one preventable cause of disability and a leading cause of death in both men and women; rapid assessment and treatment is critical to improve outcomes for patients. Guthrie Robert Packer Hospital received The joint commission primary stroke center certification in 2016, which set the following stroke measurement requirements for care of stroke patients: dysphagia screening, LDL draw and documentation, modified Rankin scale, and NIH Stroke Scale. The mission of this project is to assist Robert Packer Hospital in improving the utilization of the modified Rankin Scale, dysphagia screening, LDL screening, and NIH scale documentation at time of admission, in the management in the treatment of stroke patients. The respective benchmark goals from May 2021 to March 2021 are as follows:

1) Improvement or maintenance of 88.89% for NIH stroke scale documentation at time of admission

2) Improvement or maintenance of 97.22% for LDL documentation

3) Improvement from 83.33% to 91.66% for the modified Rankin Scale

4) Improvement from 69.44% to 76.38 % for dysphagia screening

The team received a daily list of all patients with an ICD-10 code falling under the stroke diagnosis. Starting in December 2021 until Feb 2022, 1 team member per week worked with the hospital stroke team to look over the list daily and check see which patients were not being assessed properly using our 4 measured metrics. This team member also checked to see whether patients were being placed on the hospital designated stroke pathway in EPIC. This EPIC stroke pathway has all quality metrics built in, thus increasing ease of compliance with the metrics. If a patient was not being followed up on one or more measures, the assigned team member would find the appropriate provider and message them securely on EPIC or via Guthrie email in a timely fashion, emphasizing the importance of each quality metric in improving patient outcomes. More patients were placed on the stroke pathway post-intervention, and four out of four measured metrics improved to our goal level post-intervention. Two metrics (NIHSS and LDL documentation) improved to 100%.

Audience Take Away:

- Improving modified Rankin Scale, dysphagia screening, LDL screening, and NIH scale documentation may lead to improved patient outcomes
- Proper documentation can prevent errors and ensure each stroke patient receives the appropriate workup

Biography

Rebecca Lees studied psychology with a neuroscience focus at the Pennsylvania State University and graduated with a BS in 2017. She then became an Intramural Research Training Award fellow at the National Institutes of Health/ National Eye Institute, working with Dr. Tudor Badea to research the influence of retinal ganglion cell development on visually-evoked behaviors in mice. In 2019, she began her journey as a medical student at Geisinger Commonwealth School of Medicine and is expected to graduate in 2023.

IN-PERSON

Siri Tummala
TCU School of Medicine, United States

Factors influencing patients decisions to undergo recommended spinal surgery

Spinal surgery is the final option recommended to individuals with spinal conditions when conservative treatment has failed in treating patients' symptoms. The purpose of this study is to examine the factors contributing to patients' readiness to undergo recommended spinal surgery. In this prospective study, we examined 21 patients agreeing to undergo recommended spinal fusion between August 2021 and January 2022 with a virtual questionnaire.The mean age was 61.2 years. The majority of patients were male (13 patients, 61.9%), white (19 patients, 90.5%), and privately insured (11 patients, 52.4%). Patients reported that the most important factor contributing to their interest in surgery was the severity of symptoms (14 patients, 66.7%), confidence in the neurosurgeon (4 patients, 19%), and evaluation of potential adverse outcomes (3 patients, 14.3%). Their ideal outcome in consulting with a neurosurgeon for their symptoms was surgery (16 patients, 76.2%), exercise for pain relief (2 patients, 9.5%), and being told their problem is not a neurosurgical condition (3 patients, 14.3%). There are a multitude of factors that influence patients' choice to undergo spinal surgery. These findings suggest that even though not all patients felt that surgery was their ideal outcome, all of them underwent surgery. Additional evaluations are needed to explore patients' trust in neurosurgeons' recommendations.

IN-PERSON

Gerald W. Grass
Ketamine Research Institute, Sarasota, Florida, USA

A new methodological approach to improve the "real word" effectiveness of ketamine infusion therapy for treatment-resistant depression

Objective: Ketamine is recognized as a rapidly acting antidepressant; however, discrepancies exist between the "Efficacy" reported in research studies (70-85%) versus significantly lower "Effectiveness" (18.3-45.5%) reported in community-based settings. To offset the "Efficacy-Effectiveness" gap a novel, clinically applicable methodology (RESTORE) was developed to improve both effectiveness and durability. Here we report the results of a 60-month, retrospective study of 87 patients who received RESTORE for TRD and compare outcomes to community based-studies utilizing the "standard" ketamine infusion.

Methods: Patient eligibility was determined by a three-step patient evaluation and suitability protocol. Patients received 3 infusions over 3 days and were dosed with the amount of ketamine, based on pharmacokinetic modeling, to achieve optimal blood concentrations. The medication was administered intravenously via a multimodal, variable rate infusion over 30 minutes. Following induction, patients received two additional infusions within 3-6 months before entering the maintenance phase. Symptom severity was determined utilizing the Beck Depression Index-II (BDI-II).

Results: 78 of 87 (89.4%) patients completed the 2-phase infusion protocol, of which 88.4% responded and 56.3% remitted by the fifth infusion. This compares favorably to the 18.3-45.5% response and 27.3% remission rate reported in other studies. Most notably, the average period between protocol completion and the need for maintenance infusion was 245 days compared to 26 days with ketamine infusion.

Conclusions: The novel RESTORE infusion protocol represents a clinically applicable methodological approach to ketamine infusion therapy that results in significantly improved effectiveness by 3-fold and response durability by 9-fold compared to the standard ketamine infusion.

Audience Take Away:

- What are the expected rates of response and remission following ketamine infusion therapy
- What is the therapeutic durability of ketamine therapy in treatment-resistant depression
- How to improve the overall effectiveness of ketamine infusion therapy in community-based settings
- Where to obtain additional training in the use of ketamine therapy

Biography

Dr. Grass is an Anesthesiologist who served as Assistant Professor of Anesthesiology and Pain Medicine at the Yale University School of Medicine, where the ketamine infusion for depression was developed in 1994. He was the Director of the Yale Pain Medicine Fellowship Program and the Chief of Pain Medicine for the Connecticut Veterans Healthcare Administration. Dr. Grass began utilizing ketamine in 1998 and founded the Ketamine Research Institute in 2015 to pioneer the use of ketamine therapy to treat mood disorders and chronic pain. He established the first comprehensive, international ketamine infusion training program for physicians in 2017, setting standards for the safe and effective administration of ketamine-based therapy for treatment-resistant mood disorders and other related conditions.

IN-PERSON

Maral Kasiri*[1], Terence D. Sanger[1,2,3]

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Investigation of low frequency thalamic and pallidal local field potential correlation with voluntary movement in children with dystonia

Understanding the relationship between neural activity and voluntary movement provides new insights into understanding the mechanism underlying neural control of movement in both healthy people and those with movement disorders. To investigate the relationship between deep brain oscillations and voluntary movement, we performed Deep Brain Stimulation (DBS) surgery on three patients with dystonia. Ten temporary electrodes were implanted, bilaterally, into Globus Pallidus interna (GPi), Ventralis oralis anterior/posterior (Voa/Vop), Subthalamic Nucleus (STN), Ventral Intermediate Nucleus (VIM), Pedunculopontine Nucleus (PPN), and Ventral Anterior (VA). After the surgery, the participants were asked to perform a voluntary reaching task for one minute and rest form thirty seconds, for a period of six minutes with their upper limb. We recorded Electromyogram (EMG) from the corresponding muscles and the Local Field Potential (LFP) from the contralateral side of the brain through externalized DBS leads, simultaneously, during the experiment. The LFP was high pass filtered at 1 Hz and the EMG was filtered using a nonlinear Bayesian filter to highlight the changes in muscle activity as well as the activation itself. We performed a time-frequency analysis on the LFP and explored the existence of correlation between the five subnuclei activation power in five frequency bands up to 1000 Hz with either the EMG or derivative of the EMG which is used as a measure of movement onset and offset. The preliminary results demonstrate that there exists higher correlation between the EMG and the motor-subnuclei of the deep brain regions, including GPi, VoaVop, VA, and STN compared to other regions (VIM and PPN). This correlation indicates that the motor nuclei in the brain regions activate during movement, and they are less active during the rest, regardless of the frequency bands. This study has never been done in human subjects before and the findings suggest that the low frequency deep brain recordings can potentially be a predictor of movement for neural control of prosthetics.

Audience Take Away:

- Regardless of frequency band, motor subnuclei in the human brain are active during the movement and are less active during the rest
- Motor thalamic and pallidal subnuclei activities are correlated with voluntary movement, however this correlation is indistinct in other non-motor subnuclei, like VIM
- Our results suggest that movement information is likely to be encoded in the LFP recordings regardless of the frequency. This finding shows that we can use low frequency LFP for neural control of prosthetics and movement predictions. Low frequency recording requires less computing time and processing energy

Biography

Maral Kasiri is a PhD student in Sanger lab at University of California, Irvine. She received her B.S. with honors in Biomedical Engineering from Tehran Polytechnic University in 2016 and received her M.S. in Biomedical Engineering from USC in 2018. Her focus is understanding the signal transmission in the brain during movement, in addition to exploring human motor control from information theory perspective. Additionally, she has worked on analyzing the effects of artificial sensory feedback (vibrotactile) on motor learning in children with neuromuscular disorders, specifically dystonia, and assessed the effect of sensory deficit in skill learning in these children.

IN-PERSON

Johanna Christina Reiners*[1], Laura Leopold[1], Daniela Sinske[1], Vera Hallebach[1], Michael Vogt[1], Philomena Schmidt[1], Mattia Amoroso[3], Ulrike Binder[3], Stefan O. Reber[3], Philipp Meier[1,2], Bernd KnOll[1]

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Pre-exposure to acute stress modulates the molecular response to a mild traumatic brain injury

Stress is generally defined as a real or interpreted threat to the psychological or physiological integrity of an individual that results in physiological and/or behavioral responses. Threatening stimuli trigger neuronal responses in the brain that cause an immediate adaptive systemic reaction, including the activation of (1) the hypothalamus-pituitary-adrenal axis; (2) the autonomic nervous system; (3) distinct classes of genes (e.g. immediate early genes, regeneration associated genes). Here we investigate potential interactions between two different stress stimuli, namely acute restraint stress (AS; psychological integrity) and mild Traumatic Brain Injury (TBI; physiological integrity), in an in vivo mouse model. Therefore, mice were either exposed to 45 minutes of restraint stress, followed by a mild TBI/Sham-operation; or only received a TBI/Sham-operation.

At 1h post injury, we observed, that a single exposure to AS is sufficient to dampen the gene-expression response after TBI not only in different brain regions (cortex, hippocampus, amygdala), but also in varying peripheral organs (pituitary gland, adrenal gland, heart, spleen).

Previously, the transcription factor Activating transcription factor 3 (Atf3) has been demonstrated to play a crucial role in neuroprotective functions in injury of the peripheral nervous system and in processing of stress and cellular damage in the central nervous system. Here we show, that Atf3 is a key regulator of the gene response in the pituitary gland, adrenal gland and heart, as constitutive Atf3-mutant animals neither respond to AS, TBI nor the combination of both stressors. Furthermore, Atf3KO animals showed higher mortality rates directly after TBI and more severe behavioral limitations (neurological severity score).

Overall, our data shows that a pre-exposure to AS does affect short-term outcomes after TBI.

Audience Take Away:

- The interaction and influence of two stressors of different nature
- Greater understanding of the impact of a traumatic brain injury on peripheral organs
- The role of Activating transcription factor 3 (Atf3) as an adaptive and protective factor during/following injury

Biography

Johanna Christina Reiners holds a Bachelor's degree in Molecular Life Science from Friedrich-Alexander-University Erlangen-Nürnberg (2016) and a Master's degree in Molecular and Translational Neuroscience from Ulm University (2019). In late 2019 she started her PhD in the laboratory of Prof. Dr. Knöll, where she is currently working on the interaction between acute psychological and physical stress.

IN-PERSON

Judith Stefanie Schlett*, Bernd Baumann, Thomas Wirth
Institute of Physiological Chemistry, Ulm University, Ulm, Germany

NF- κB is a critical mediator of age-dependent white matter loss

Inflammaging represents an accepted concept where the immune system shifts to a low-grade chronic pro- inflammatory state without overt infection upon aging. In the CNS, inflammaging is mainly driven by glia cells and associated with neurodegenerative processes. White matter degeneration is a well-described process in the aging brain which manifests in myelin loss finally resulting in neurological deficits. However, understanding of the underlying molecular mechanisms remains limited. Oligodendrocytes and their precursor cells are responsible for production, homeostasis and maintenance of the myelin sheaths. Mature oligodendrocytes are highly energy demanding cells due to their unique functions, and thus highly sensitive to different forms of stress. So far it is open how altered inflammatory conditions like inflammaging affect homeostasis and function of oligodendrocytes. We investigated the role of NF-κB, a well-known mediator of inflammatory and stress responses, in the homeostatic control of oligodendrocytes using a conditional gain-of-function mouse model allowing oligodendrocyte-specific activation of IKK/NF-κB signaling (IKK2-CA$^{PLP\text{-}CreERT2}$) in the adult organism. Chronic NF-κB activation in mature oligodendrocytes was sufficient to initiate overall neuroinflammatory conditions in the CNS accompanied by motoric and neurological deficits which progress with age. Ultrastructural analyses revealed degeneration of the corpus callosum as well as loss of myelin sheaths and accordingly, myelin proteins were found decreased in IKK2-CA$^{PLP\text{-}CreERT2}$ mice. Interestingly, RNA-Seq analysis of isolated primary oligodendrocytes revealed gene expression signatures pointing to NF-κB mediated Integrated Stress Response (IRS), Senescence Associated Secretory Phenotype (SASP) and Post Mmitotic Cellular Senescence (PoMiCS) appearing prior to white matter loss. Taken together, our data indicate that IKK/NF-κB signaling, a central effector of diverse stress responses, is able to trigger PoMiCS of mature oligodendrocytes thereby disturbing myelin maintenance and finally forcing aging-dependent white matter loss.

Audience Take Away:

- Selective NF-κB activation in mature oligodendrocytes is able to initiate global neuroinflammation in the brain
- Post-mitotic cellular senescence of mature oligodendrocytes is critically regulated by NF-κB/ inflammaging
- Integrated stress responses and cellular senescence are important mediators of white matter loss
- Cell-type-specific interference with NF-κB function or downstream effectors offer new therapeutic strategies to attenuate age-dependent white matter degeneration

Biography

Judith Stefanie Scheller graduated with a degree in Food Chemistry and Toxicology in 2016 from the Karlsruhe Institute of Technology. Afterwards she worked 2 years on protein misfolding and amyloidosis before she joined the group of Prof. Dr. Thomas Wirth to obtain her PhD within the excellence programme of the International Graduate School of Molecular Medicine at Ulm University. She is about to finish her PhD working on inflammation driven neurodegeneration.

IN-PERSON

R Smaili*[1], R Belfkih[2], M Bourkia[1]

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Imatinib as a treatment for prion disease: A historical case of sporadic creutzfeldt jacob disease complicating chronic myeloid leukemia on imatinib

Objective: To describe the first case of sporadic Creutzfeldt Jakob disease (CJD) in a patient with chronic myeloid leukemia who survived more than two years in imatinib mesylate treatment.

Method: A 60-year-old female followed for Bcr- Abl positive chronic myeloid leukemia in imatinib. Who presented psychiatric disorders for which was put on antipsychotic medication for 12 months, with a progressive onset of dementia and extrapyramidal syndrome of the upper limb. A brain CT scan, metabolic assessment and vitamin dosages were normal.

The patient was put on L-dopa in addition to psychiatric drugs for more than 6 months, with initial improvement and then progressive worsening of the extrapyramidal syndrome, associated with myoclonus, visual disturbance. MRI Brain was performed and demonstrated bilateral symmetrical hyper signals of the striatum. The EEG showed a theta rhythm and triphasic periodic abnormalities. A CSF test for 14.3.3 was positive. A diagnosis of probable sporadic CJD was made. Imatinib mesylate was majored to 800mg/day in addition to symptomatic treatment. Patient died 8 months later.

Results: Over two years of survival-term in a patient with sCJD and Bcr-Abl positive chronic myeloid leukemia on imatinib mesylate treatment.

Conclusions: The Bcr-Abl pathway inhibitors represent a promising treatment in CJD. But more clinical studies are needed to establish their role in long-survival sporadic CJD patients.

Keywords: Sporadic CJD – Chronic myeloid leukemia – BcrAbl pathway – Imatinib mesylate

Biography

Dr. Smaili is a resident doctor in the Department of Internal Medicine and Clinical Immunology, under the direction of Professor Bourkia at the University Hospital of Tangier-Tetouan-AL Hocima, Morocco. He is preparing his master's degree in connective tissue diseases at the University of Lille, France. He is also preparing a specialization degree in Internal Medicine at the University of Paris, France, for one full year. He graduated with a medical degree in 2016 from the Faculty of Medicine of Oujda, Morocco, and has worked as an emergency physician for two years afterwards.

IN-PERSON

Lisa Yu[1], Govinda Sharma[1], Dhananjaya D.[1], Sarah G. Cook[2], Kaveh Matinkhoo[1], Glynnis E. Jensen[1], David J. Press[1], Jessica Bik-Jing Lee[1], Ye Cai[1], Jonathan Gallant[1], Stanley K. A. Opare[1], Xue Chen[1], Jing Li[1], Limei Chang[1], Francesca Maule[3], Joseph E. Tucker[1,2], Jillian M. Hagel[1], Sheetal A. Raithatha[1], Peter J. Facchini*[1,3]

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Design and synthesis of novel psilocin prodrugs with improved metabolic and pharmacokinetic properties as candidate therapies for treatment-resistant anxiety disorders

The psychedelic compound psilocybin has shown therapeutic benefit in the treatment of several psychiatric diseases. A recent randomized clinical trial conducted at Johns Hopkins Bayview Medical Center demonstrated the efficacy of psilocybin-assisted therapy in the treatment of Major Depressive Disorder (MDD). Similarly, recent phase IIb data from a large study evaluating psilocybin therapy for treatment-resistant depression presented statistically meaningful and long-term reduction in depressive symptoms. Despite these compelling clinical results, concerns regarding the duration of the psychedelic experience produced by psilocybin pose a significant barrier to its widespread therapeutic application. Psilocybin is the naturally occurring prodrug of the neuroactive compound psilocin. When orally administered, exposure to the acidic gastrointestinal environment together with enzymatic processing by intestinal and hepatic alkaline phosphatases lead to the dephosphorylation of psilocybin producing elevated levels of systemic psilocin. These plasma levels are measurably detectable up to 24 hours and produce a psychoactive episode lasting as long as 6 hours post-ingestion. In an effort to positively modify the kinetics of the acute psychedelic response, we have engineered a library of novel prodrug derivatives of psilocin, producing a diversity of established metabolically amenable moieties substituted at the 4-carbon position of the core indole ring. This library consists of thirty unique compounds represented by nine distinct prodrug classes. Each molecule was screened in vitro for metabolic stability using isolated human serum, and human cellular fractions derived from liver and intestinal tissues. This screen revealed fifteen molecules that produced measurable levels of psilocin in vitro, with ester and carbonate-based prodrug derivatives significantly represented. These fifteen molecules were further evaluated for pharmacokinetic (PK) profiles in mice, assessing plasma exposures of both residual prodrug and resultant psilocin. PK results confirmed the efficiency of ester and carbonate-based prodrug metabolism upon oral and intravenous administration, achieving approximately one-tenth the maximum plasma concentration of psilocybin-derived psilocin and comparable 8-hour exposures at each relative dose. Of note, all novel prodrugs tested maintained significantly reduced overall exposure, with no measurable levels detected at 24hrs post-dose. Finally, all novel prodrug molecules were screened for bioactive potential, by evaluating 5-HT2A receptor stimulation in vivo using the established behavioural marker of Head Twitch Response (HTR) in healthy mice. Strikingly, five prodrug derivatives produced peak HTRs that approached or exceeded levels induced by an equivalent dose of psilocybin. Of these active compounds, two ester-based prodrug molecules produced long-term anxiolytic benefit in chronically stressed mice evaluated in the marble burying psychiatric model. Overall, this screening campaign identified novel candidate prodrugs of psilocin with altered metabolic profiles and significantly reduced pharmacological exposure, potentially attenuating the duration of the psychedelic response. These molecules still maintained the long-term psychiatric and physiological benefits characteristic of psilocybin therapy. Additionally, these modified parameters also offer the opportunity for altered routes of administration bypassing conventional oral dosing.

Audience Take Away:

- Natural psychedelics such as psilocybin can be altered and improved through ProDrug strategies
- Pharmacokinetic (PK) parameters can be altered through rational drug design
- Altered PK parameters translates into in vivo behavioral changes in mice using established head-twitch response (HTR) model
- Screening campaigns can identify novel candidate prodrugs of psilocin with altered metabolic profiles and significantly reduced pharmacological exposure
- Screening campaigns potentially attenuate the duration of the psychedelic response

Biography

Dr. Peter Facchini is the Chief Innovation Officer at Enveric Biosciences Inc. and a professor of biochemistry at the University of Calgary. He has held the Canada Research Chair in Metabolic Processes Biotechnology and has received awards for his achievements in innovation and entrepreneurship as the co-founder of four private and two publicly traded biotechnology companies. In addition to his work on psychedelics at Enveric Biosciences, his research has involved the complete elucidation of several medicinally important metabolic pathways, including the biosynthesis of morphine in opium poppy, and the reconstitution of these pathways in microorganisms.

IN-PERSON

Xue Chen[1], Jing Li[1], Lisa Yu[1], Govinda Sharma[1], Dhananjaya D.[1], Francesca Maule[2], Sarah G. Cook[1], Stanley K. A. Opare[1], Jonathan Gallant[1], Sheetal A. Raithatha[1], Jillian M. Hagel*[1], Peter J. Facchini[1,2]

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[2]Department of Biological Sciences, University of Calgary, Calgary, Alberta, Canada
[2]Neurology department, University Hospital of Tangier, Morocco

Isolation of a novel N-methyltransferase from psychedelic cane toad (*Rhinella Murina*) deployable in bio-based production platforms for psychiatric drug discovery

The skin and paratoid gland secretions of many toads in the Bufonidae family contain several natural products with importance to traditional medicine. Some of these compounds, such the indole amines 5-hydroxy-N,N-dimethyltryptamine (bufotenine) and 5-methoxy-N,N-dimethyltryptamine (5-MeO-DMT) are psychedelics with therapeutic potential for the treatment of psychiatric mood disorders. The availability of novel biosynthetic enzymes acting on tryptamine derivatives would facilitate synthetic biology efforts toward biocatalytic and microbial-based production of novel, psychedelic-inspired pharmaceuticals. We mined the transcriptome of cane toad (*Rhinella murina*) to isolate a novel N-methyltransferase (RmNMT) homologue distantly related (40-45% amino acid sequence identity) to mammalian indolethylamine N-methyltransferase (INMT) and nicotinamide N-methyltransferase (NNMT). Steady-state enzyme kinetic characterization showed that RmNMT coverts tryptamine and serotonin to N,N-dimethyltryptamine (DMT) and bufotenine, respectively, in a two-step reaction. RmNMT also catalyzes the robust N-methylation of a wide variety of other tryptamine derivatives, although indolethylamines substituted at 7- and 2-positions were poor substrates or were not accepted, respectively. Batch-fed *in vivo* conversions in *Escherichia coli* cultures followed by extraction chemistry yielded a pairwise collection of natural and novel primary and N-methylated indole amines in sufficient quantity and purity for pharmacological assessment. Compounds were assayed for 5-HT_{2A} and 5-HT_{1A} receptor-binding activities to investigate the impact of N-methylation on differentially substituted tryptamine derivatives with previously known or potential effects on psychiatric mood. K_i values of N,N-dimethylated tryptamines were approximately 2-5 fold higher than their N-desmethyl counterparts suggesting reduced binding affinity to 5-HT_{1A}. However, timed exposure to MAO (Monamine Oxidase)-rich Human Liver Microsomes (HLMs) revealed reduced the stability of most N-desmethyl tryptamines, in support of a broad protective function of N-methylation in vivo. Results revealed key structure-activity relationship features informing future drug design efforts.

Audience Take Away:

- Psychedelic drugs are expected to be represent novel treatments for mood psychiatric disorders
- Existing psychedelics can be improved or built-upon to inspire a new collection of pharmaceuticals available to patients recalcitrant to current treatments
- We use synthetic biology to build psychedelic-inspired drugs; here, we present the example of using natural sources (Toad) to find & develop new tools useful for drug development
- Audience will observe an expansive pipeline to develop psychedelic-inspired drugs, from gene mining in Toad to synthetic biology to pharmaceutical testing and ultimately *in vivo* models

Biography

Dr. Hagel currently holds the role of Vice President Innovation with Enveric Biosciences, Inc. (NASDAQ: ENVB). She completed her Ph.D. in 2010 at the University of Calgary, Canada, in the field of specialized plant metabolism and opiate biochemistry. While working as a post-doctoral fellow on a multi-laboratory Genome Canada project aimed at sourcing new genes from medicinal plants, Dr. Hagel co-founded three private and two publicly traded biotechnology companies. Over her career, she has published over 40 peer-reviewed articles, largely in the field of natural products and associated biochemistry.

IN-PERSON

Ilhan Yoo, MD
Department of Neurology, Nowon Eulji Medical Center, Eulji University School of Medicine, Seoul, Republic of Korea

Parsonage–turner syndrome following COVID-19 vaccination: Case series

Background: Severe acute respiratory syndrome coronavirus 2 is a coronavirus that has recently appeared, rapidly spreads, and results in coronavirus disease 2019 (COVID-19). Moderna mRNA-1273 and Pfizer-BioNTech BNT162b2 vaccines are among the most widely used and effective vaccines that play a key role in preventing COVID-19 infection. However, the safety of COVID-19 vaccines has not been fully elucidated. To date, various neurological adverse events post-vaccination have been reported. This report aimed to describe six patients who developed Parsonage–Turner syndrome after Moderna mRNA-1273 and Pfizer-BioNTech BNT162b2 vaccination without underlying predisposing conditions.

Methods: This report consists of six cases of PTS following COVID-19 vaccination, who visited the Nowon Eulji Medical Center in Seoul, South Korea, from August 13, 2021 to January 20, 2022.

Results: Magnetic resonance imaging and electrophysiologic studies revealed enlargement of the brachial plexus and neuropathy involving the medial antebrachial cutaneous nerve or C5, C6 trunks of the brachial plexus, respectively. After treatment of most patients with high-dose oral corticosteroids, their symptoms, including shoulder pain, weakness, and neuropathic pain, gradually improved.

Conclusions: Early treatment with high-dose corticosteroids is recommended to improve the outcomes of patients with Parsonage-Turner syndrome after COVID-19 vaccination. Large prospective studies are needed to further prove the causal relationship between COVID-19 vaccination and Parsonage–Turner syndrome.

Audience Take Away:

- If patient will complain weakness and shoulder pain at uni- or bilateral limbs after COVID19 vaccination, physicians should consider possibility of brachial plexitis associated with COVID19 vaccination
- Brachial plexitis following COVID19 vaccination could well treated with high dose oral corticosteroid
- Brachial plexitis could develop immediately or several days after COVID19 vaccination

Biography

Dr. Ilhan Yoo studied medicine at the Ajou University, South Korea and graduated as MD in 2011. He then entered Chunag Ang university hospital, South Korea, and trained as neurologist. He received his master degree in 2019 at the Chung Ang university. After one year fellowship supervised by Dr Jung-Joon Sung at the Seoul National University hospital, South Korea, he obtained the position of an Assitant Professor at the Nowon Eulji University hospital. He has published 2 articles in SCI(E) journals.

IN-PERSON

Min Sung Gee*[1], Seung Hwan Son*, Seung Ho Jeon[1], Jimin Do[2], Namkwon Kim[3], Yeon-Joo Ju[1], Soo Jin Lee[1], Eun Kyoung Chung[1], Kyung-Soo Inn[3], Nam-Jung Kim[1,3], Jong Kil Lee[1]

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A selective p38α/β mapk inhibitor alleviates neuropathology and cognitive impairment, and modulates microglia function in 5xFAD mouse

Chronic neuroinflammation, aggressive Amyloid Beta (Aβ) deposition, neuronal cell loss and cognitive impairment are pathological symptoms of Alzheimer's disease (AD). Regarding these symptoms, resolution of neuroinflammation and inhibition of Aβ-driven pathology might be a novel strategy for AD therapy. Efforts to prevent AD progression have identified that p38 Mitogen-Activated Protein Kinase (MAPK) is a promising target for AD therapy. However, the actual therapeutic effect of selective p38 MAPK inhibitors in AD has not been ascertained yet. In this study, we explored the therapeutic potential of NJK14047, a selective p38 MAPK inhibitor, using an Alzheimer's disease mouse model, 5xFAD. The mice were injected 2.5 mg/kg NJK14047 or vehicle every other day for 3 months. Morris water maze task and histological imaging analysis were performed. Protein and mRNA expression levels were measured using immunoblotting and qRT-PCR. In *in vitro* studies, the cytotoxicity of microglial conditioned medium and astrocyte conditioned medium on primary neurons were measured using MTT assay and TUNEL assay. NJK14047 treatment downregulated phospho-p38 MAPK levels, decreased the amount of Aβ deposits, and improved spatial learning memory in 5xFAD mice. Interestingly, these effects were associated with the decrease of inflammatory responses and the increase of the phenotype markers of alternatively activated microglia which were effective for phagocytosis and degradation of Aβ peptides. Furthermore, NJK14047 treatment reduced the number of Fluoro-jade B positive cells, a class of degenerating neurons, in the brains of 5xFAD mice. The neuroprotective effect of NJK14047, achieved via the restoration of microglia function, was further confirmed by in vitro studies.

Audience Take Away:

- Importance of neuroinflammation and glial phenotypes in Alzheimer's Disease (AD)
- The experimental procedures and tips for AD research using 5xFAD mouse
- The experimental procedures and tips for neuroinflammation research using primary neurons and glial cells
- Mouse behavior test procedures and tips
- Morris water maze test
- Novel objective recognition test
- Y-maze test
- Passive avoidance test

Biography

Mr. Gee studied Biology and Pharmacology at the KyungHee University, Korea and graduated as B.S. in 2017. He then joined the research group of Prof. Lee at the KyungHee University to major in neurodegenerative disorders especially Alzheimer's disease. He has published 9 research articles in SCI(E) journals, and 3 of them, he participated as the first author. He is currently preparing 3 other research articles and planned to receive the PhD degree in 2023.

IN-PERSON

Vildan Tunçbilek*[1], Bengü Altunan[1], Rıdvan Mercan[2], Aysun Unal[1]
[1]Department of Neurology, Tekirdağ Namık Kemal University Faculty of Medicine, Tekirdağ, Turkey
[2]Department of Internal Medicine, Section of Rheumatology, Tekirdağ Namık Kemal University Faculty of Medicine, Tekirdağ, Turkey

Multiple autoimmune syndrome: Combination of neuromyelitis optica spectrum disorder with myastenia gravis, systemic lupus erythamatosis and hashimato tiroiditis

Autoimmunity reflects an altered immune status, therefore the presence of more than one disorder is not uncommon. The coexistence of three or more autoimmune diseases in a patient constitutes Multiple Autoimmune Syndrome (MAS). This is an interesting case of a middle-aged female in whom clinical signs of NMOSD and MG co-occurred and got the diagnosis of SLE and thyroiditis during her evaluation.

A 31-year-old female patient was admitted to our clinic with complaints of diplopia, severe nausea-vomiting, balance disorder, fever, and widespread muscle aches.Family history was unremarkable for any autoimmune disorder. She was a diagnosed case of hashimato tiroiditis. MRI examination revealed a T2 hyperintense lesion extending from the bulbus to the posterior pons. In the examinations, Aq4 antibody was positive, Antinuclear Antibodies (ANA) test was strongly positive. Laboratory workup showed normal complete blood counts, markedly elevated transaminases and alkaline phosphates. During the follow-up period, acetylcholine receptor antibody was positive, which was examined due to fluctuating eye findings, nazone speech, and dysphagia. High fever, evoloemic hyponatremia was evaluated as central origin. Pericardial effusion detected in cardiac examination and pleural effusion observed in thorax Bt were associated with rheumatologic picture. In addition to high-dose pulse steroid and plasmapheresis treatments, multidisciplinary treatments were arranged. The patient, whose rituximab treatment was started, is followed up without clinical worsening and disability in the two-year follow-up.

Patients with diagnosis of NMOSD need special attention as multiple immune-mediated disorders may be present simultaneously or sequentially during the course of the disease process. These patients need close surveillance for the development of another autoimmune disease, so as to control the current disease and to prevent future complications.

Audience Take Away:

- Neuromyelitis Optica Spectrum Disorders is an autoimmune inflammatory demyelinating disease of the central nervous system.
- There have been many reports on its association with other disorders including systemic and organ specific autoimmune diseases. However, in the literature it is rare that all of these co-existing autoimmune diseases.
- This case report emphasizes the importance of a multidisciplinary team approach for a better understanding of disorders related to the breakdown of immune tolerance.
- İt is important that of adequate immunological education with clinical information for positive future outcomes and patient management

Biography

Dr. Vildan Tunçbilek graduated from Gulhane Faculty of Medicine, University of Health Sciences in 2018. She then worked as a general practitioner in a short time in Department of Emergency, Marmaraereğlisi District State Hospital. After that, she started her neurology residency training as a Research Assistant Doctor at Tekirdağ Namık Kemal University, Department of Neurology in 2019. Still, continuing specialization training. She has 3 national, 2 international presentation. She published 3 articles; one of them editorial and 2 of them research articles.

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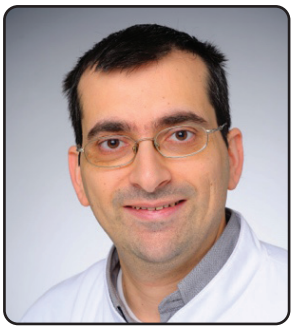
VIRTUAL



Georgios Matis MD, MSc, PhD, FINR (CH)
University of Cologne, Faculty of Medicine and University Hospital Cologne,
Department of Stereotactic & Functional Neurosurgery Cologne, Germany

Which is the role of ziconotide in the intrathecal pain treatment?

Ziconotide is a synthetic, water-soluble cone snail venom-derived peptide with a molecular weight of 2,639 Daltons. It is a nonopioid analgesic that selectively binds to N-type voltage-sensitive calcium channels on primary nociceptive afferent nerves in the dorsal horn of the spinal cord. This mechanism releases analgesic neurotransmitters into the synaptic gap and subsequently blocks pain signal transmission. Ziconotide does not easily cross the blood-brain barrier, instead revealing its highly potent antinociceptive effect only after intrathecal administration. Because it has a narrow therapeutic window, careful dose titration, and a lag time to allow for onset (and offset) of analgesia and adverse effects are required. The presentation will focus on a recently published consensus proposal and highlight the potential of this drug as well as the areas where additional experience is needed.

Audience Take Away:

- Expand the knowledge on possible neuromodulation therapies
- Learn how intrathecal therapy can help patients with chronic pain
- Learn how a non-opioid drug (Ziconotide) could be a viable treatment option
- Learn about the advantages and disadvantages of Ziconotide
- Help physicians provide one more therapy to their chronic pain patients

Biography

Dr. Georgios Matis is a senior consultant for neurosurgery. He leads the chronic pain / spasticity sector of the Department of Stereotactic & Functional Neurosurgery in the University Hospital of Cologne. He has been trained in Greece (General University Hospital of Alexandroupolis, G. Papanikolaou General Hospital of Thessaloniki & 417 Army Equity Fund Hospital of Athens), USA (Department of Neurosurgery, Weill Cornell Medical College, New York, NY), Switzerland (Department of Neuroradiology, University Hospital of Zurich, Zurich) and Germany (Department of Stereotactic & Functional Neurosurgery, University Hospital Cologne, Cologne). Dr. Matis is a member of two medical associations (Thessaloniki, Greece & North Rhine, Germany) and also a member of the German Neuromodulation Society (DGNM) and the International Neuromodulation Society (INS). He serves as reviewer for many international journals and is Editorial Board member for Neuromodulation: Technology at the Neural Interface and Interventional Pain Medicine and Neuromodulation. He holds the position of Editor-in-Chief of the Internet Journal of Neurosurgery. Dr. Matis has published many articles in Greek and international Pubmed-indexed journals and hold many lectures as invited speaker in numerous international congresses and webinars. At the same time, he is Public Education Committee member of the International Neuromodulation Society. Dr. Matis is involved in many international clinical studies and has been active as instructor for many colleagues in Germany and abroad. He is also an active member of the medical advisory board of the German CRPS Support Group and member of several online consultation platforms. He is actively involved in social media trying to raise awareness about spinal cord stimulation and neuromodulation.

VIRTUAL

Brandon Lucke Wold MD, PhD, MCTS
Department of Neurosurgery, University of Florida

Recent advances in the understanding of the pathophysiology of neurotrauma

Traumatic brain injury is an important concern in the realm of neurological diseases. At present, our understanding of TBI and its downstream effects both acutely and chronically is limited, although in the past decade, many recent advancements have presented. Current treatment regimens have largely followed academic society guidelines with a special emphasis on intensive care unit management. Its diverse etiology in disease presentation has lent its way to difficulty in seeing promising advancements succeed in clinical development, although our understanding of this disease course and the therapeutic targets currently in development continues to grow. These areas of interest include excitotoxicity, oxidative stress, blood brain barrier disruption, neuroinflammation, and white matter degeneration. At present, emerging treatments have included cell-based therapies and the neuroprotective agents focused on recovery following inflammatory damage. The current review highlights in detail the advancements in our understanding in the advancements in our understanding in the pathophysiology of TBI, the current treatment regimens, and the emerging treatments in development.

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 honors from Baylor University. He completed his MD/PhD, 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, the pharmaceutical company CTE cure, and was a science advocate on Capitol Hill through the Washington Fellow's program. He has also served as president of the WVU chapters for the American Association of Pharmaceutical Scientists, Neurosurgery Interest group, and 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 is wife. Brandon also enjoys reading, playing piano, discussing philosophy, and playing chess. He is currently a Pgy5 neurosurgery resident at University of Florida with pursuing endovascular enfolded training and was awarded the Dempsey Cerebrovascular Research Fellowship.

Leya Maliekal*[1], Brandon Askar[1], Jessica Moore[1], Marc Feldman[2]
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Treating rubral tremors with divalproex sodium

Rubral tremors, or Holmes' tremors, are a type of movement disorder that appear post-cerebellar/thalamic injury. Optimal treatment approaches for rubral tremors are still being understood and include pharmacological options such as dopamine precursors, anticonvulsants, and anticholinergics, as well as deep brain stimulation of thalamic nuclei. Our patient was a 72-year old man who presented to the E.D. for evaluation after new-onset unilateral jerking and tremors, two months after a hemorrhagic cerebrovascular accident with craniotomy and residual deficits including right-sided upper extremity hyperflexion & hemineglect, right-sided lower extremity motor weakness, and aphasia. The tremors were visibly present in his right lower extremity, occurring every 2-3 seconds with a sharp amplitude of 3-5 inches, present at rest and worsening with intentional movement. The tremors were not visibly present in his right upper extremity, likely due to stiffness from previous CVA deficits, but tremors were easily felt through palpation. Our patient was previously able to ambulate on his own; however, after onset of these tremors, he was no longer able to ambulate. Our patient was first treated with levetiracetam, to which he reported minimal improvement in tremor frequency, amplitude, and pain. However, tremors were still observed. Levetiracetam was discontinued, and our patient was started on divalproex sodium. He reported resolution of symptoms within 24 hours, with continued improvement persisting over the next week. The right lower extremity tremors were no longer visibly present and were only mildly present upon palpation, occurring every 5-6 seconds with a pulsing sensation. Our patient reported immense relief in pain and discomfort and was able to safely ambulate with assistance. Per this case, divalproex sodium has shown to be highly effective in resolving symptoms of rubral tremors. No pharmacological agent has been formally identified as a first-line agent for rubral tremors, with most references typically suggesting levadopa for treatment. In our patient, divalproex sodium was chosen over levadopa to avoid the large side effect profile of levadopa. Because divalproex sodium was so efficacious in our patient, we suggest that further study would be beneficial to understand the safety and generalizability of divalproex sodium treatment for other cases of rubral tremors, especially when compared with other pharmacological agents.

Audience Take Away:

- Minimal cases of rubral tremor resolution have been described in literature, most of which describe effectiveness of levodopa and/or deep brain stimulation. Only 2-3 case reports specifically describe benefits of valproic acid therapy. This abstract offers more evidence for the effectiveness of divalproex sodium, allowing a more comprehensive understanding and contribution to the library of pharmacological treatments available for rubral tremors
- Levodopa and deep brain stimulation include many side effects, such as dizziness, forgetfulness, and confusion, as well as the risks of surgical procedures. Divalproex sodium allows efficacious therapy with minimal side effects. As a result, it would be worthwhile to compare the effectiveness of levodopa, deep brain stimulation, and divalproex sodium in order to maximize the efficacy of therapy while decreasing side effects
- This abstract can raise awareness that divalproex sodium is an efficacious agent in treating rubral tremors

- Further study of divalproex sodium can contribute to shaping guidelines for treatment for rubral tremor treatment within an in-patient setting, providing physicians with evidence-based information to properly care for their patients
- This abstract also opens up many other avenues regarding exploring the pharmacological effects and functions of divalproex sodium within the human body, specifically with movement disorders

Biography

Leya Maliekal is a third year medical student at Wayne State University School of Medicine in Detroit, MI and previously completed her BS in Public Health at Wayne State University. Brandon Askar is a third year medical student at Wayne State University School of Medicine in Detroit, MI and previously completed his BS of Cell Molecular Biology at the University of Michigan. Jessica Moore is a second year Physician Assistant student at Wayne State University in Detroit, MI and previously completed her Bachelor of Science in Nursing at Eastern Michigan University.

VIRTUAL

Flavia I Spiroiu
McMaster University, Canada

The association between intolerance of uncertainty with social interaction anxiety and performance anxiety - mediating factors

Background: Despite the potential importance of Intolerance of Uncertainty (IU) as a transdiagnostic feature that may contribute specifically to social anxiety, empirical data on the construct has been relatively scant and focused largely on Generalized Anxiety Disorder (GAD) and Obsessive Compulsive Disorder (OCD). A number of studies have indicated that IU and social anxiety are highly associated; however, the question of what intermediate variables may help explain this association requires exploration. Identification of potential indirect effects, such as interpretations of ambiguous social information and sensitivity to real or perceived negative evaluation, is thus warranted.

Method: Sixty-six undergraduate students from Lakehead University ranging in age from 17 to 52 completed the study. Participants were administered the Intolerance of Uncertainty Scale (IUS), the Social Interaction Anxiety Scale (SIAS), the Social Phobia Scale (SPS); a measure assessing fear of being scrutinized in specific performance situations), and the Brief Fear of Negative Evaluation Scale - 2 (BFNE-2). They moreover completed two measures that assessed negative interpretation bias in social anxiety, namely the Ambiguous Judgment Questionnaire (AJQ) and the Ambiguous Social Situation Interpretation Questionnaire (ASSIQ).

Results: Mediation models were estimated using a bootstrapping approach (Hayes, 2013). The first analysis indicated that fear of negative evaluation significantly mediated the relationship between IU and social interaction anxiety (*ab* = .24), as well as the relationship between IU and performance anxiety (*ab* = .24). Negative interpretations of ambiguous social information (as reflected by ASSIQ scores) significantly mediated the relationship between IU and social interaction anxiety, (*ab* = .09). Negative interpretations of ambiguous interpersonal events on the AJQ likewise significantly mediated the relationship between IU and social interaction anxiety (*ab* = .05).

Conclusion: Findings suggest that IU about the possibility of being negatively judged by others in interpersonal or performance situations may lead to a fear of such negative evaluation, which may in turn induce and maintain social anxiety. Moreover, individuals with social interaction anxiety may be so distressed by uncertainty about the possible meaning or consequences of ambiguous social information, that they may biasedly interpret the information negatively in hopes of experiencing a swift reduction in distress. Indeed, this is consistent with prior research indicating that individuals with GAD were so distressed by uncertainty that they preferred a certain negative outcome to an uncertain outcome (Koerner & Dugas, 2006). Future research would therefore benefit from examining whether and how targeted reductions in fear of negative evaluation and negative interpretations of ambiguous social information can ameliorate social anxiety symptoms.

Biography

Flavia Spiroiu obtained her Bachelor of Arts in Psychology at Ryerson University in Toronto, Canada. Ms. Spiroiu subsequently earned her Master of Arts in Clinical Psychology from Lakehead University in Thunder Bay, Canada. She is currently conducting her Ph.D. in Clinical and Health Neuroscience at McMaster University and the Anxiety Treatment and Research Centre in Hamilton, Canada under the supervision of Dr. Noam Soreni. Miss Spiroiu's research examines the cognitive and behavioral mechanisms of transcranial direct current stimulation (tDCS) in the context of cognitive behavioral therapy (CBT) and exposure and response prevention (ERP) for obsessive-compulsive disorder (OCD).

David Chang
NHS Hull University Teaching Trust, United Kingdom

VTE prophylaxis in stroke

Background: Venous ThromboEmbolism (VTE) prophylaxis is a recognised part of stroke management in European Stroke Organisation (ESO) guidelines, and a component in the NICE Stroke pathway. The practicalities of the prescribing and application of these modalities whether pharmacological or mechanical differs between organisations, and is dependent on the local stroke service.

Aim: This work audited the practice of VTE prophylaxis in Stroke patients in a large UK tertiary centre and Stroke service provider against recommendations from NICE and ESO guidelines, to identify if the right VTE prophylaxis modalities were being prescribed and effectively given, and what barriers could exist to prevent these being effectively employed.

Methods: Data was collected from 2 weeks of inpatient admissions to the local Stroke Unit. Clinical notes, online admission forms and drug records were reviewed to identify the VTE prophylaxis modality prescribed and given, taking into account stroke pathology and decisions on VTE prophylaxis with clinical information such as preexisting anticoagulation, whether a patient was thrombolysed, mobility and falls risk. The patient was also examined to see if mechanical prophylaxis such as intermittent pneumatic compression(IPC) devices were active on the patient. Data was collated, formatted and analysed with Microsoft Excel

Results: Sample size of 51 patients. 50 patients had confirmed stroke on imaging 18% (9/51) hemorrhagic strokes, 78% (39/51) ischaemic strokes, 2% (1/51) mixed stroke, with the remaining one patient found to have an uncomplicated left intracranial aneurysm. 84% of patients (42/51) had appropriate VTE prophylaxis prescribed in the form of IPC devices or Low Molecular weight Heparin (LMWH) after clinical consideration, or had no VTE prophylaxis after an appropriate clinical assessment taking mobility and falls risk in account. Of the 8/51(16%) remaining patients, VTE prophylaxis deviated from guidelines, with

Thrombo-Embolic Deterrent (TED) Stockings or inappropriate LMWH or a combination thereof or no VTE prophylaxis prescribed. It was noted that a high number of patients who had VTE prophylaxis prescribed did not have them on or did not have them turned on - 26% (13/51) on admission, 14.3% (7/51) by 72 hours. There were however no associations with VTE incidences, bleeds or 30-day mortality within the limits of this audit.

Conclusions: Following a local clinical governance meeting, it was found that most deviations from Stroke VTE prophylaxis guidelines happened due to patients moving from a medical admissions unit to the Stroke unit, where general medical junior doctors may not have been aware of stroke guidelines. With regards to IPC devices being prescribed but not being active on patients, it was highlighted that after physiotherapy or occupational therapy assessments, it was not clear who would be reattaching detached IPC devices after patients were returned to the bedside which was a potential source of deviation. The logistics of having IPC devices available in a general medical unit were also highlighted as Stroke unit beds may not always be available.

Audience Take Away:

- It is important to identify if there are structures in place for ensuring patients with stroke have access to IPC devices should there be no Stroke Unit availability
- Physiotherapy and Occupational therapy sessions during which patients have IPC devices unattached and potentially left unattached can impact on the provision of VTE prophylaxis to stroke patients
- Local education for junior doctors outside of the Stroke unit on VTE prophylaxis in stroke versus the general inpatient population can be important for the overall provision of Stroke care

Biography

Dr. Chang obtained his medical degree from the University of Edinburgh, Scotland in 2017, continuing medical training in the United Kingdom National Health Service and completing Foundation Year training in Glasgow, Scotland. He commenced Acute Care Common Stem (ACCS) Acute Medicine broad based training in NHS England, obtaining the MRCP (London) Diploma from the Royal College of Physicians of the United Kingdom in 2021, and is currently in his penultimate year of ACCS training in Hull, UK.

Tebboth AM*, Mohamed K
Department of Neurology, Eastbourne District General Hospital, East Sussex Healthcare NHS Trust, UK

Cerebral Amyloid Angiopathy Related Inflammation (CAARI) diagnosis and response to intravenous methylprednisolone in a district general hospital

Introduction: Cerebral Amyloid Angiopathy Related Inflammation (CAARI) is a rare but now increasingly recognised presentation of Cerebral Amyloid Angiopathy (CAA). The clinical presentation is most commonly an acute or subacute onset of cognitive decline, rapidly progressive dementia, personality changes, headache, seizures, focal neurological deficits and confusion.

Case presentation: A 78-year-old man, with a history of prostate adenocarcinoma and coronary artery bypass graft, presented to hospital with a fall. He experienced 6 months of rapidly progressive impaired working, visuospatial and semantic memory. He suffered with significant lapses in concentration with a remarkably low attention span and significant hallucinations. On the ward, he was markedly confused, agitated and aggressive. Inflammatory markers were normal, as were his renal function, liver function, bone profile and full blood count. A lumbar puncture showed slightly raised proteins. MRI head showed pronounced subcortical, central white matter oedema with multiple micro-hemorrhages. With all this combined, using the chung criteria, we diagnosed the patient with CAARI. The facility of performing a brain biopsy was not available to confirm the diagnosis. He was then treated with methylprednisolone 1000mg IV for 3 days followed by prednisolone 60mg reducing the dose every week by 10mg until discontinue. Not only did his MRI oedema decrease, but his mental state showed significant improvement as well. His hallucinations resolved, and his attentiveness and concentration improved.

Conclusion: This will help to highlight ways in which to diagnose CAARI to those who do not have the facility to perform a brain biopsy or in patients in which is it inapplicable. We focus specifically on the history, mental state examination and radiology. Steroid therapy led to significant improvements clinically and radiologically.

Audience Take Away:

- They will learn about a rare complication of cerebral amyloid angiopathy in the form of Cerebral Amyloid Angiopathy-Related Inflammation (CAARI)
- How CAARI can present and what investigations are needed to rule out other causes
- How CAARI can be diagnosed in a hospital where some investigations are not available
- How CAARI can be treated and the response to this treatment

Biography

Dr Alice Tebboth studied Medicine at Cambridge University and graduated with MB BChir in 2019. She then started her Foundation Year Training at Brighton and Sussex Hospitals NHS Trust and then East Sussex Healthcare NHS Trust. She is continuing her medical training in Eastbourne with a specific interest in neurology.

VIRTUAL

Flavia Spiroiu*, Noam Soreni
Department of Psychiatry & Behavioural Neurosciences, McMaster University, Hamilton, ON, Canada

Cognitive and behavioral mechanisms of transcranial direct current stimulation in obsessive-compulsive disorder

Background: Obsessive-Compulsive Disorder (OCD) is a common and severe psychiatric disorder. Cognitive Behavior Therapy (CBT) with Exposure and Response Prevention (ERP) is the gold standard intervention for OCD, but more than 40% of patients with this disorder remain impaired even after treatment. Transcranial Direct Current Modulation (tDCS) is a safe and minimally invasive procedure that modulates cortical function by delivering a weak electrical current over the scalp. Although a growing body of literature suggests that tDCS may potentially improve CBT outcomes in OCD, a knowledge gap remains concerning the specific cognitive and behavioural mechanisms by which tDCS may augment the outcomes of CBT for OCD.

Purpose: The purpose of this study, which is in progress, is to investigate the cognitive and behavioural mechanisms of action of tDCS in the context of CBT with ERP for OCD. Specifically, it examines whether a single active session of bilateral tDCS is associated with 1) improved decision-making in uncertain conditions and 2) increased willingness to engage in ERP.

Methods: The target sample size is 50 adult patients diagnosed with OCD (N = 25 Active, 25 Sham), who undergo a course of group CBT for OCD at the Anxiety Treatment and Research Clinic (ATRC) in Ontario, Canada. Participants complete baseline measures prior to administration of the 20-minute tDCS. OCD symptom severity is assessed by the Yale-Brown Obsessive Compulsive Scale (Y-BOCS) and willingness to engage in ERP is measured by an Exposure Feedback Form (EFF). The study follows a double-blind placebo (sham) controlled design. Cathodal and anodal electrode placement is over areas F3 (left Dorsolateral Prefrontal Cortex [DLPFC]) and F4 (right DLPFC), respectively. In both active and sham conditions, the current increases from 0 to 2mA over a period of 30s. To measure immediate cognitive effects, the Iowa Gambling Task (IGT)-a measure of decision-making under uncertainty and the cognitive outcome variable in the study-commences 10 minutes into the tDCS session. Upon completion of the tDCS and IGT, participants once again complete the behavioral outcome measure of their willingness to engage in ERP (i.e., the EFF). The EFF is repeated two days after the tDCS.

Data Analyses/Results: The *cognitive* hypothesis (IGT) will be tested using a one-way, two-group ANCOVA with Group (Active vs. Sham) as the between-subjects factor and covariates being any group baseline difference. The *behavioural* hypothesis (engagement in ERP) will be analyzed using a 2x2 repeated measures ANCOVA with time as the within-subjects factor, group as the between-subjects factor, and the same covariates.

Progress: Twenty-three participants have been tested to date. tDCS was well tolerated by all participants, and no adverse events were reported or observed. The acceptability of the procedure was high, as all participants completed the tDCS and all study assessments with no missing data. Potential implications. Evidence of tDCS effects on cognitive and behavioural aspects of ERP can provide a compelling rationale for the subsequent development and administration of a clinical trial of tDCS efficacy in OCD.

Audience Take Away:

- Overall, the audience will learn that tDCS is not only a putative candidate for adjuvant therapy for OCD and a range of neuropsychiatric conditions, but also that it is a valuable tool in neuroscience research, as its focality can be used to explore several brain aspects
- It has been established that successful ERP is associated with functional changes in OCD-related brain circuits. Findings from this ongoing study present a promising avenue of investigation, namely the link between brain circuits implicated in OCD, their associated neurocognitive domains, and how the deficits in these domains affect treatment response
- One of the most consistently identified areas of cognitive dysfunction in OCD is decision-making, particularly under uncertain conditions. Given that ERP involves situations that patients invariably perceive as uncertain, tDCS may improve decision-making processes in the context of such conditions. In turn, having improved decision-making in uncertain situations may also act to improve engagement in ERP, with regards to challenging exposure practices. The more an individual engages in exposure practices, the greater the treatment effects
- This research could serve as preliminary groundwork for future researchers who may wish to examine changes in a much broader range of cognitive functions associated with tDCS administration in the context of CBT/ERP for OCD patients. In this respect, tDCS can be employed to modulate specific regions of the orbitofronto-striato-pallida-thalamic network, cortico-striatal-thalamo-cortical (CSTC) pathway, fronto-parietal network, and/or other extended regions to detect cognitive effects on attention, executive function, language, learning and memory, perceptual-motor function, and social cognition
- In sum, researchers, health practitioners, and laypeople will learn that tDCS has unique characteristics such as ability to induce antagonistic effects in cortical excitability according to the parameters of stimulation; concomitant use with neuropsychological tests; non-invasiveness and thus absence of pharmacokinetics interactions—a putative substitutive/augmentative agent in neuropsychiatry; and low-cost and portability, making it suitable for increasing access to novel therapies for patients with OCD and other neuropsychiatric disorders

Biography

Flavia Spiroiu obtained her Bachelor of Arts in Psychology at Ryerson University in Toronto, Canada. Ms. Spiroiu subsequently earned her Master of Arts in Clinical Psychology from Lakehead University in Thunder Bay, Canada. She is currently conducting her Ph.D. in Clinical and Health Neuroscience at McMaster University and the Anxiety Treatment and Research Centre in Hamilton, Canada under the supervision of Dr. Noam Soreni. Miss Spiroiu's research examines the cognitive and behavioral mechanisms of transcranial direct current stimulation (tDCS) in the context of cognitive behavioral therapy (CBT) and exposure and response prevention (ERP) for obsessive-compulsive disorder (OCD).

VIRTUAL

Cristian Ravariu*[1,2], Mihai Popescu[1,3]

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[3]Division of IT Devices & Service of "Carol Davila" Medicine and Pharmacy University Bucharest, B- dul Eroii Sanitari nr, Bucuresti, Romania

Systemic models: From the neuromuscular junction to the upper levels of the nevrax

To make possible a systemic methodology for the treatment of the neuromuscular pathology, some models must be developed to predict what stimulus can optimal interfere with the system function in order to modify its state. Stripped muscles are part of somatic system - the interaction system of human body with the environment. Their rigorous control is one of the main nervous system objective. This paper presents few models of the process control for the different levels of interfaces between the human nervous system and locomotion system. A systemic approach of the nervous system, at the control engineering level and the integration of the means of treatment and investigation as scheme control block permits an integrative approach of treatment or rehabilitation processes. The proposed models associate some electronic functions to different biological parts, benefiting on the system theory for the human body. From cellular level, the neuron is behaving like a comparator with signal integration on inputs. Depending on the membrane specific capacity C_m and equivalent conductance G_m, the input current density is processed accordingly to the switch time between the steady state potential and action potential transitions - excellent modeled by electronic comparators. It must be taking into account that the spatial and temporal summation of the stimulus, each one being either an excitation or inhibition, is coming up from multiple synapses, in a short period of time. Then, step by step, more portions of neuronal membrane are associated with systemic blocks that depict an entire cell. The next step is to model the sensitive neurons. They work like voltage controlled oscillators, because every sensation is converted in a voltage level on the input resulting a voltage pulses train at the output. On the other hand, according with the recent work in the domain of cellular and molecular neurobiology, based on Henman principle, a neuronal pole consists in all the motor neurons provided for the command of a single muscle. The recruitment order in the situation of a command that arrives from the upper level of nevrax, occurs after their order of magnitude: the motor units with slow fibers first, then the motor units with medium fibers and the motor units with fast fibers at last. Finally, three control loops for the upper levels of the nevrax were identified and modeled by a Process - Controller continuous loop.

Audience Take Away:

- The audience will be able to apply these models in order to develop useful tools in neuromuscular diseases
- People working in the field of medical recovery in locomotors system can use these models based on systemic approach, able to depict the body reactions to external stimulus, like reflexology
- This research work can be extended to other faculties in the bioengineering field, to expand their research or teaching area from neuro-locomotion system to a systemic approach

Biography

C. Ravariu studied Microelectronics and Bioelectronics at the Polytechnic University of Bucharest, Romania. He graduated as MS in 1993. He worked as scientific researcher first 5 years at Institute of Microtechnology, Bucharest, then joined the Polytechnic University of Bucharest. After multiple foreign stages in Bio-Nano-Engineering (Patras, Greece), Nano-devices (EPFL, Switzerland), Organic Electronics (LAAS, France), he received Post Doc degree in 2012 in Romania. Since 2013 he obtained the position of Full Professor at the Polytechnic University of Bucharest, Faculty of Electronics. He has published more than 250 research articles. Since 2014 he is Chairman of Romanian IEEE Electron Devices Chapter.

VIRTUAL

Harinder Jaseja
Physiology, NIMS University, Jaipur, India

Optimizing deep brain stimulation parameters in intractable epilepsy: An EEG based innovative approach

Deep Brain Stimulation (DBS) of Anterior Thalamic Nucleus (ATN) has established as an effective adjunctive therapy for patients with Intractable Epilepsy (IE) not suitable for epilepsy brain surgery and/or vagal nerve stimulation. The judicious selection of DBS Parameters (DBSPs) plays a crucial role in the success of ATN-DBS. Conventionally, DBSPs are selected by trial and error requiring multiple sessions and hospital visits warranting a strong need for optimization of the DBSPs with objective assessment of its effects. The author presents an EEG-guided novel and superior approach to the selection of effective DBSPs targeted to induce EEG-desynchronization, which is known to exert potent antiepileptic influence with possibly possession of an additional anti-kindling effect that can suppress or even arrest the ongoing process of epileptogenesis in the patients with intractable epilepsy in addition to exercising control over the intractable seizures. It is further claimed that the innovative EEG-guided approach can successfully optimize the DBSPs resulting in (a) minimum sessions of DBSP adjustments, thereby reducing the frequency of hospital visits (b) minimum side effects and (c) minimum consumption of the device battery; thus, prolonging its life. Preliminary results of the clinical application of the novel approach in the selection of the DBSPs in a small case series have been very promising and encouraging despite which it is strongly recommended that well designed large sized studies are required for its validation and successful clinical outcome.

Audience Take Away:

- Deep Brain Stimulation (DBS) is an emerging and effective therapy for patients with intractable epilepsy that constitute about one-third of all patients with epilepsy
- The faculty dealing with patients with epilepsy will be able to use this knowledge not only in their research and teaching, but also in their clinical practice
- This technique will certainly provide the audience-clinicians an edge over their counterparts indulged in similar practice and/or teaching and research
- The novel technique will not only enhance the efficacy of treatment of the patients but also would reduce the cost and time of patients

Biography

Dr Harinder Jaseja has worked as Professor in Physiology in G R Medical College, India and is presently Professor in Physiology, NIMS University, Jaipur, India. He was ranked Second in Epilepsy Research in India in a 2013-published national journal. He has discovered a novel target (pedunculopontine nucleus) for deep brain stimulation in intractable epilepsy and an innovative approach to the selection of anterior thalamic nucleus deep brain stimulation parameters in patients with intractable epilepsy. He has also published novel guidelines for management of patients with cerebral palsy He has published more than 75 international papers and is member of several editorial boards.

VIRTUAL

Kunal Bhanot*, Muhesh Kumar Taheem*, Tony Goldstone, Steve Gentleman

Department of Brain Sciences, Imperial College London, London, United Kingdom

Investigating the effects of blast traumatic brain injury on the hypothalamus in a porcine model

The increasing prevalence of blast Traumatic Brain Injury (bTBI) in military personnel has stimulated further research into this field. The neuroendocrine system can be severely affected in blast survivors and anterior pituitary dysfunction has been characterised in soldiers returning from service. Imaging studies have failed to report structural changes associated with hypopituitarism, which has prompted further investigation into hypothalamic disturbance in blast models. This study utilised a porcine model of bTBI to examine structural changes in the hypothalamus between bTBI animals (n=2) and non-bTBI (n=4) controls. Changes in microglial activation around the hypothalamus and differences in Growth Hormone Releasing Hormone (GHRH) expression were also examined.

Animals were categorised into blast (n=2), sham (n=2) and control (n=2) cases. Blast animals were subjected to an overpressure wave and systemic haemorrhage. Sham animals were only subject to systemic haemorrhage and control animals had no intervention. Animals were sacrificed 4 hours after intervention. Haematoxylin and eosin staining was used to examine structural differences. Anti-Iba1 and anti-GHRH immunohistochemistry was used to observe differences in microglial activation and GHRH expression, respectively. Ventricular deformities such as parenchymal compression were noted in blast cases alone. There was evidence of sub-ependymal oedema in both blast and sham cases compared to control cases. Microglial activation was increased in both blast and sham cases compared to control cases. Furthermore, GHRH immune reactivity was greatest in the porcine arcuate and paraventricular nucleus, however differential expression of GHRH could not be determined.

Evidence of structural damage to the hypothalamus was noted in bTBI animals and could provide a basis on which to explore hypothalamic damage in blast survivors. However, the pattern of neuroinflammation observed was likely attributable to systemic haemorrhage rather than blast exposure. Finally, anatomical landmarks such as the anterior fornix were associated with the localisation of GHRH secreting neurons in the pig.

Audience Take Away:

- Consider anterior pituitary dysfunction in blast injury survivors
- Hypothalamic damage may be due to both blast exposure and ischaemia in blast injury survivors
- Structural damage to midline structures is more prominent in traumatic blast injury, especially at phase interface such as the ventricular surface
- Neuroinflammation in blast injury is likely multifactorial but tackling this early could be an avenue in preventing long term hypothalamic dysfunction

Biography

Dr Kunal Bhanot is currently a military doctor based in Colchester, UK. He intercalated in Neuroscience during medical school at Imperial College and has an interest in blast injuries.

Dr Muhesh Kumar Taheem is currently a surgical trainee in London, UK. He too intercalated in Neuroscience and also has extensive research experience in stroke medicine, having explored the effects of therapeutic cooling.

VIRTUAL

Paul Raj
Department of Psychology, Jyoti Nivas College Autonomous, Bengaluru, Karnataka, India

Reading and reading disorders in children: perspectives from developmental neuroscience

Reading is an important skill for mastery in social relationships, academic performance, emotional regulation and most types of work. Reading impairments, one of the most common neurodevelopmental disorders affecting approximately 5-10% of school-going children across languages, interfere with their ability to read and diminish how they learn to read which can persist and compound into adulthood. The onset and developmental course of reading disorders are largely unknown, even though neuroimaging studies suggest atypical brain maturation. The growing empirical evidence advocates that the neural pathways underlying the reader's perceptual, cognitive and linguistic processes that pertain to mastery of reading vary with age. Therefore, these changing processes may affect adversely the neurocognitive characteristics of reading disorders in children at each developmental stage.

The presentation will synthesize empirical findings from developmental neuroscience to increase our understanding of distinctive neural pathways underlying reading and reading disorders. The presentation will begin with an overview of the brain processes fundamental to typical and atypical reading. In the process, the focus will be given to the results of studies that have employed neuroimaging approaches. Second, through the emphasis on scientific attention to developmental effects and synthesizing empirical findings from cross-sectional studies on reading disorders at various ages, the presentation will expand the knowledge base of reading disorders in children. The etiology of reading disorders can be better studied by segregating between primary and secondary impairments that unfold along with the development. Finally, the presentation will portray empirical findings from existing longitudinal studies that study developmental reading pathways beginning in the preliterate stage at both group and individual levels. Such an attempt can contribute to the accuracy of early identification and enable targeted intervention of deficits in foundational pre-literacy skills and reading fluency, leading to improved outcomes in at-risk or affected populations.

Biography

Paul Raj is an Assistant Professor of Psychology at Jyoti Nivas College Autonomous, Bengaluru, India and teaches courses in Basic Psychological Processes, Lifespan Development, Cognitive Psychology, and Positive Psychology, apart from guest lectures in Counselling Skills and Learning Disabilities. He has obtained MSc in Counselling Psychology and M.Phil. in Learning Disabilities. His PhD topic is on Cognitive Functioning and Mathematical Abilities among Primary School Children and his research interests include child and adolescent development, neuropsychology, specific learning disorders and positive psychology. He has published two books, two book chapters and four articles in reputed journals.

VIRTUAL

Maria Joao Sacadura
Catholic University of Portugal and University of Coimbra, Coimbra, Portugal

Adherence to treatment regimen in end-stage renal disease. Insights from neuroscience of "habit" and the importance of social networks in case management

The present communication proposes are question how the family and the community are structuring actors in the management of chronic kidney disease; questions the habits and routines of a chronic renal patient based on the neurosciences approach and relates this information to cognitive deficts referred by patients in a survey applied in a PhD social work research in Portugal. Relationships with the family environment, with institutions and with the resource system are the object of study of the social work both to understand the situation of difficulty and to modify the causes that determine it. (Adapt. Carvalho, cit. Campanini, 2015, p.1). Social work can have a prominent paper in supporting patients and families to develop good habits and routines to manage the disease. Seidel et al. (2014, p. 628), referred that due to limited daily life activities, cognitive deficits are not always evident for the patients' social environments, (...) but they have direct relevance for daily function in CKD patients. Is it possible that decreased attention and concentration, memory deficits, reduced mental alertness and executive functions affect patients in end-stage renal disease. Among patients with terminal CKD receiving hemodialysis, more than 85 % had cognitive deficits (Murray et al. 2006).

Social work as a case manager can be more aware of these interfaces and support more effective interventions with families, resource systems and with the multidisciplinary team of the dialyses unit. Transpose these inferences to the social assessment and intervention, contributes to generate new perspectives and ways to improve intervention and to look for the best participation and patient decision-making concerning adherence to treatment regimen.

Audience Take Away:

- To give an applied perspective of the relevant paper of family and community networks in the phenomena of adherence to treatment in the context of end-stage renal disease
- To reflect on how these contributions may or may not inform the practice of social intervention with people on a hemodialysis program
- The analysis will entail a correlation between the neuroscience of habits, social work, and neurology in end-stage renal disease in adherence to treatment regime

Biography

Graduate and PhD student in Social Work. Worked at Fresenius Medical Care Portugal (FMC), both as a Director and Social Worker and had been a Member of the Ethics Committee of FMC Portugal – Nephro Care. Postgraduate in Social Gerontology; attendance of the Business Management Program with specialization in Human Resources; advanced training in Approaches and Strategies for the Intervention in Complex Social Problems, among others. Experience in the development of pioneering projects co-financed by the EU in the field of reconciling family and work life. Joint author of the study "Ageing of the Portuguese Population: Dependency, Activation and Quality", mandated by Economic and Social Committee.

VIRTUAL

Manuel Narvaez Pelaez*[1,2], Marina Mirchandani-Duque[1], Miguel A. Barbancho1, Alexander Lopez-Salas[1], Jose Erik Alvarez-Contino[1], Natalia García-Casares[1], Kjell Fuxe[2], Dasiel O. Borroto-Escuela[1,2,3]

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[2]Department of Neuroscience, Karolinska Institute, Stockholm, Sweden
[3]Department of Biomolecular Science, Section of Physiology, University of Urbino, Urbino, Italy

New preclinical findings in depression and neurodegenerative diseases: Role for galanin and neuropeptide y interaction in the hippocampus

Accumulating evidence for Neuropeptide Y (NPY) and Galanin (GAL) interaction was shown in various limbic system regions at molecular, cellular and behavioral-specific levels. Dysregulation of hippocampal neurogenesis is linked to several neurodegenerative diseases and depression, where boosting hippocampal neurogenesis in these patients emerges as a potential therapeutic approach. The purpose of the current work was to evaluate the role of NPY and GAL interaction in the neurogenic actions on the dorsal and ventral hippocampus. We studied the Y1R agonist and GAL effects on: hippocampal cell proliferation through the Proliferating Cell Nuclear Antigen (PCNA); the expression of neuroprotective and anti-apoptotic factors and the survival of neurons and neurite outgrowth on hippocampal neuronal cells. The functional outcome was evaluated in the object-in-Place task and the forced swimming test. We demonstrated that the Y1R agonist and GAL and promote cell proliferation and the induction of neuroprotective factors. These effects were mediated by the interaction of NPYY1 (Y1R) and GAL2 (GALR2) receptors, which mediate the increased survival and neurites outgrowth observed on neuronal hippocampal cells. These cellular effects are linked to the improved spatial-memory effects after the Y1R agonist and GAL coinjection at 24 hours in the object-in-place task and in the forced swimming test. Our results suggest the development of heterobivalent agonist pharmacophores, targeting Y1R-GALR2 heterocomplexes, therefore acting on the neuronal precursor cells of the DG in the dorsal hippocampus for the novel therapy of neurodegenerative cognitive-affecting and depressive diseases.

Audience Take Away:

- Understanding Neuropeptide Y and GAL interaction through Y1R-GALR2 heteroreceptor complex
- How the Y1R agonist and GAL may promote cell proliferation in the DG of the dorsal and ventral hippocampus and the induction of neuroprotective factors, such as BDNF and Bcl-2
- How Y1R-GALR2 heteroreceptor complexes mediate survival and neurites outgrowth on neuronal hippocampal cells
- How these cellular effects may be linked to spatial-memory and antidepressant effects
- The development of heterobivalent agonist pharmacophores, targeting Y1R-GALR2 heterocomplexes, therefore acting on the neuronal precursor cells of the DG in the dorsal and ventral hippocampus for the novel therapy of neurodegenerative cognitive-affecting and depressive diseases

Biography

Manuel Narváez cursed Medicine and surgery degree in 2005, with the best academic record of his promotion, in 2006 I obtained a competitive pre-doctoral excellence scholarship from the Andalusian board. The research activity developed allowed him to carry out 5 months visits during 2009 and 2010 at the Karolinska Institute in Stockholm to obtain the European mention. In 2012 I obtained the European PhD thesis with cum laude qualification, the extraordinary PhD award from the faculty of medicine, thesis prize from medical college of Malaga (2012) and the prize from college of pharmacists of Malaga (2013). Up to 6 postdoctoral visits to the Karolinska Institute in Stockholm collaborating on multiple research projects establishing collaborative links with the Swedish research group, during the years 2012-2021, total more than 1 year. The research results have been published successively in congresses of international and national relevance. In addition, innovative articles have been published, including in the first quartile of impact index in its category and with quality indices, including high cite numbers. Our team has performed multidisciplinary research and worked in a highly integrative manner at different systems levels, we have contributed to the GPCR receptor-receptor interactions field focus in CNS diseases, such as depression, Parkinson, addiction drugs and Alzheimer.

VIRTUAL

Irene Fasciani*, Francesco Petragnano, Francesco Colaianni, Gabriella Aloisi, Roberto Maggio and Mario Rossi

Department of Biotechnological and Applied, Clinical Sciences, University of L'Aquila

Detrimental effects of light at 610 nm on dopamine neurons and implications for PD onset

Light pollution has recently become a significant global environmental issue. Interestingly, several in vivo approaches and worldwide correlation studies of Parkinson disease onsets have shown to strongly correlate with the distribution of light pollution. In particular it has been shown that artificial light had profound detrimental effects on dopaminergic neurons. In fact, the prolonged exposure of rats and mice to fluorescent light resulted in increase of neuromelanin granules and damage of dopamine neurons in the substantia nigra. In fact, these findings suggest that light pollution may be a key environmental factor implicated in the preferential degeneration of dopamine neurons in PD. In addition, we have recently shown that electromagnetic wavelengths around 600nm, also emitted by florescent light lamps, reach deep into the mouse, rat, human brains.

This project aims at characterizing light-induced neurodegeneration by using the dopaminergic cell model: MN9D. This cell line was exposed to four wavelengths: 485 nm, 535 nm, 610 nm and 710 nm in order to evaluate the wavelengths at which the fluorescent light was responsible for the detrimental effects observed on the dopaminergic neurons of rats and mice used in our previous studies. In particular we showed that light at 610 nm was the most harmful wavelength for the MN9D and stickily had no effect on control non-dopaminergic cells. Moreover, 610 nm exposure of differentiated MN9D caused a decrease in cell viability and a strong statistically significant increase in ROS production compared to controls, especially when cells were co-treated with an oxidative agent (H2O2). Our results strongly suggest that light potentiates oxidative stress specifically in dopaminergic cells through dopamine oxidation. Moreover, future experiments will be performed using human iPSC-cells from different patients having mutations associated with PD to investigate the relationship between environment such as the prolonged exposure to artificial light and genetic factors.

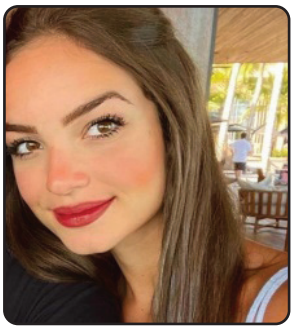
Audience Take Away:

- That artificial light had profound detrimental effects on dopaminergic neurons, in vivo.
- That the light wavelength at 610 nm reaches deep inside mouse, rat and human brains.
- That chronic and acute light exposure at 610 nm of the dopaminergic neuronal cell model, MN9D results in increases of ROS production and overall neuronal cytotoxicity.
- In conclusion, the presented data would strongly suggest that chronic artificial light exposure might become a risk factor for Parkinson's Disease.

Biography

Dr. Irene Fasciani studied Biology at University of L'Aquila, Italy and graduated in 2012. She then joined the research group of Professor Roberto Maggio at the same Institute and received her PhD degree in 2016. During 5 years of post-doctoral fellowship, she worked on molecular aspects of neurological disorders and inflammation. She published more than 20 articles in scientific journals.

VIRTUAL



Julia Souza E Costa*, Bruany Antoniolli Bianchi, Mario Vicente Campos Guimaraes, Ana Beatriz Lima Pedroza, Natalia Moreno Coelho, Josue Andrade Martins

Anhembi Morumbi University, Sao Paulo - SP, Brazil

Victim of trauma trauma by high caliber firegun: Case report

Background: The main reason for neurological disability and death in young adults is Traumatic Brain Injury (TBI), affecting about 60 million people annually. Recovery is directed by the intensity and severity of the lesion, as well as by the dimension of subsequent damage to inflammatory and neuroendocrine responses, therefore, in the management of patients should minimize the occurrence of secondary reactions.

Objective: This paper aims to investigate and clarify the consequences of TBI and the possible managements that contribute to a better patient outcome.

Methods: The information contained in the description of the clinical case was obtained through a review of the available medical file and an interview with the patient. To elucidate what was described; data obtained from a collection of scientific articles on TBI were used.

Results: The medical records of a 51-year-old military police sergeant with traumatic brain injury at the Military Police Hospital of the State of Sao Paulo were obtained. In this document, it is said that the trauma occurred during sector patrols, where he was shot bilaterally in the frontal cranial region with two shots of 7.62mm caliber. The patient was rescued by the fire department vehicle and the Rescue and Emergency Care Group (GRAU) at the scene of the trauma, where he was intubated, sedated and received mechanical ventilation. In the hospital, he was evaluated and taken to the surgical center by the neurosurgery team, where he was submitted to a broad bilateral craniectomy (bilateral fronto-temporo-parietal), superior sagittal sinus closure, hemostasis and duroplasty. The patient evolved to minimally conscious state in the late postoperative period and was subsequently submitted to cranioplasty with prosthesis made by prototyping with good results.

Conclusion: The traumatic brain injury caused by firearms is a real and growing possibility in the clinical experience in this scenario of urban violence, being necessary to pay attention to the possible damages caused by the loss of the vascular brain self-regulation.

In this case, it was found that the most aggressive and immediate treatment allowed a recovery of the patient, confirming the findings in the maintain this approach contributes to a reduction in mortality.

Due to the addition of neurodegenerative processes resulting from the expansion of the metabolic activity of neurons stimulated by the death of neurons affected in trauma, leading to a significant increase in free radicals, oxidative stress and neuroinflammatory cascade, one of the events that gains more and more significance in TBI is excitotoxidade. Despite the patient's postoperative success, we understand that TBI has social and economic consequences due to post-traumatic stress, such as impairment of executive function, impulsivity, aggressive behavior, depression, among others, it is imperative to stimulate preventive measures. In addition, it is a noble and sensitive organ injury, for this reason, the training of professionals in the trauma and hospital scene is essential for the patient to have a systematic and rapid monitoring and evaluation to avoid this tragic outcome.

Audience Take Away:

- The main reason for neurological disability and death in young adults is Traumatic Brain Injury (TBI), affecting about 60 million people annually.
- Among the causes that lead to higher mortality rates in penetrating TBI are injuries from firearms.
- The prognosis of these patients depends on the type of projectile, distance from the shot, medical assistance provided at the site of the occurrence and, especially, the place of entry into the projectile. Recovery is directed by the intensity and severity of the lesion, as well as by the dimension of subsequent damage to inflammatory and neuroendocrine responses, therefore, in the management of patients should minimize the occurrence of secondary reactions.
- The traumatic brain injury caused by firearms is a real and growing possibility in the clinical experience in this scenario of urban violence, being necessary to pay attention to the possible damages caused by the loss of the vascular brain self-regulation.
- In situations of homeostasis, the regulation of cerebral vasculature is maintained by mechanisms. However, this is lost in the TBI scenario, and these peripheral blood pressure changes have as consequences cerebral edema, hemorrhagic progression and even evolution to intracranial hypertension.
- It is known that TBI may come from primary or secondary lesions. In primaries, the brain is affected during impact on the skull, leading to bone fractures, neuronal and vascular damage. In secondary lesions, what occurs is a compromise of the blood-brain barrier, neutrophilic invasion and microglial activation.

Biography

Medical student at the Anhembi Morumbi University. Researcher at the University of Sao Paulo (USP) - Department of Immunology. Research grant from FAPESP for the project "Elimination of RASSF9 expression in B16F0 melanoma cell line by CRISPR/Cas9 technology". Chapter Publisher about transcranial stimulation in neuropsychiatry diseases. Researcher in Institute of Memory, Group studies of Neurosciences ONG - Doctors of the World, Oncogenetics and Oncogenomics, and Infectology. Lastest article accepted for publication “Letter: Importance of Cobalt-60 Dose Rate and Biologically Effective Dose on Local Control for Intracranial Meningiomas Treated with Stereotactic Radiosurgery. NEUROSURGERY, 2022”. She has interests in Genetics, Immunology, Immunotherapy, Precision Medicine, Neurosurgery, Neuroimmunology, Neurology.

VIRTUAL

Ana Beatriz Lima Pedroza*, Mario Vicente Campos Guimaraes, Bruany Antoniolli Bianchi, Natália Moreno Coelho, Josue Andrade Martins, Julia Souza e Costa
Anhembi Morumbi University, Sao Paulo – SP, Brazil

The role of microsurgery on the treatment of meningioma: A case report and literature review

Background: Meningiomas are the most common intracranial tumors, they can occur on any intracranial or spinal dural surfaces and even with benign predominance, are related to neurological deficits and decreased quality of life.

Objectives: This paper aims to demonstrate the importance of recent technologies in the treatment of brain tumors, focusing on meningiomas and how it impacts the patient's life.

Methods: The case reported in this article was directed by contemplating the patient's medical records, being discussed and organized from the most recent literature.

Clinical Presentation: A 52-years-old male relates dizziness, worsening by movement, and headache in the frontal region of the skull, grade 6 in pain scale, which improved by dipyrone administration. The patient was previously diagnosed with hypertension and dyslipidemia. First, the patient was diagnosed with labyrinthitis. Three months later, the symptoms had worsened. At physical examination, he presented proportionated spastic hypertonia, hyper reflexive hemiparesis on the right side. The computerized tomography showed temporoparietal expansive lesions on the right side, possibly from the meningothelial coat. The microsurgery approach was chosen for treatment. The patients evolved with no post-surgical complications nor neurological deficits and resolution of the symptoms.

Results: Meningiomas come from arachnoid meningolian cells, most of these tumors appear intracranially, but may also have incidence along the spinal cord, they tend to be benign and grow slowly and gradually, sometimes emerging in inaccessible places.

Meningiomas have symptoms common to other tumors, such as headache, seizures, psychosis, memory disorders, diplopia and anxiety, which may delay diagnosis. In addition, meningiomas located in the right temporoparietal region are associated with depressive symptoms, being able to develop symptoms of acute schizophrenia if there is no intervention. Although intracranial neoplasms are more frequent, the scarcity of studies makes molecular profile analysis difficult for individualized therapeutic choices. Then today, in clinical practice, the most widely used method for the diagnosis of meningioma is Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) to assess the stage and thus the severity of the disease.

Therefore, to assist in the choice of management, the World Health Organization (WHO) commonly classifies them as: benign (grade I), atypical (grade II) and malignant (grade III). Although most meningiomas are benign in nature, only incidental and asymptomatic meningiomas are usually accompanied by radiological surveillance, for growing and symptomatic tumors microsurgery is the gold standard treatment due to its efficacy and if residual tumor tissue it is treated with radiotherapy.

Thereby, the microsurgery is a microscopic technique that has advanced over the years, making it effective and safe, in addition to demonstrating a significant increase in the rate of total tumor removal and decreasing postoperative recurrences.

Conclusion: Meningiomas are the most frequent tumors in the nervous system and are commonly benign. Due to the lack of data from the practice of molecular analysis, the diagnosis is mostly performed by imaging, CT and MRI. Thus, allowing to evaluate whether the type of conduct will be expectant, surgical with or without adjuvant treatment, guaranteeing the safety and quality of life of the patient.

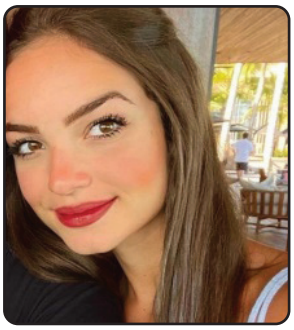
Audience Take Away:

- Meningiomas are the most common intracranial tumors that require new surgical and therapeutic techniques at all times. Bringing better prognosis and quality of life to patients, in addition to contributing to other cranial tumor treatments.

Biography

Ana Beatriz Lima Pedroza, medical student in Anhembi MorumbiUniversity, a researcher in scientific initiation by the São Paulo State Research Support Foundation through a research in the area of immunometabolism since 2022 by the University of São Paulo. Publication in the chapter "Duchenne Muscular Dystrophy - An Integrative Review at Pauster in 2022. Approved for an exchange in Portugal, for the project “Era Uma Vez Brasil” in 2016. Volunteer chemistry monitor during high school. Anhembi Morumbi University Dermatological, Oncology and Genetic group studies member.

VIRTUAL



Julia Souza E Costa*, Bruany Antoniolli Bianchi, Mario Vicente Campos Guimaraes, Ana Beatriz Lima Pedroza, Natalia Moreno Coelho, Josue Andrade Martins

Anhembi Morumbi University, Sao Paulo - SP, Brazil

Case Report - Facial nerve palsy associated with COVID-19 infection

The number of cases reporting the frequent impact on the nervous system due to SARS-CoV-2 infection is increasing and raising justifiable concerns. This impact, usually manifested in more severe cases of the disease, can lead to the most diverse neurological disorders. Among the causes of these neurological sequelae, the affinity for the ACE2 receptors of the virus, the neurotrophic potential, the recurrent infection of astrocytes, pericytes, macrophages and the malfunction of the immune system stand out. The latter involves an exacerbated immune system response, triggering a cytokine storm, as well as an autoimmune reaction. Facial nerve palsy appeared in several case reports after the presentation of the common symptoms of the disease and subsequent confirmation of diagnosis with medical exams. Bearing that in mind, our objective was to report the case of a patient who developed bilateral facial nerve palsy to evidence this potential manifestation of COVID-19. The medical records of a 46-year-old patient without comorbidities with Bilateral Facial Palsy at the Hospital were obtained. In that document, it is stated that the described paralysis occurred seven days after the onset of symptoms of COVID-19. Concomitantly, the patient presented paresthesia in four limbs, dysarthria and dyspnea. After performing tests, he was diagnosed with bilateral VII cranial (facial) palsy, secondary to SARS-CoV-2 infection, and then started therapy with human immunoglobulin, responding positively to treatment. SARS-CoV-2 can manifest itself in several ways, focusing on respiratory symptoms. However, the considerable increase in exposed cases of damage to the neurological system caused by this infection confirms that the investigation and deepening of the subject is stimulated, since the high affinity with ACE2, its viral replication in the nasal mucosa and the triggering of the cytokine storm can lead to serious complications to the patient, leading to death. Neurological disorders are a real possibility in the clinical experience of the new coronavirus pandemic, and it is necessary to pay attention to the possible damage caused by hypoxia and the inflammatory processes of the disease, especially in risk group patients. Under the exposed case, it was verified that the use of human immunoglobulin reduced the severity of the current disease of the infectious processes, allowing a partial or total recovery of the clinical picture of facial paralysis in a short period of time. Despite the success, considering the etiology of the paralysis and the physiology of SARS-CoV-2 in the nervous system and the patient's history, the need for further studies to elucidate all the mechanisms involved in the clinical manifestations caused by this infection is unquestionable, so that new effective treatments can be developed, as well as to facilitate the identification of the most advisable therapy in each case, in order that damages are mitigated.

Audience Take Away:

- In view of the dimension of the pandemic caused by the new coronavirus and the uniqueness of its mechanism of action, which makes it capable of infecting several systems, in addition to the Respiratory System, neurological sequelae cannot be ruled out. In view of the above, we verified a diversity of reports of patients who had Nervous System involvement and had from milder symptoms, such as headaches, to more serious and of great concern, such as stroke, meningo encephalitis, acute myelitis, guillain- barre syndrome, among others

- Neurological disorders subsequent to the disease were reported during the worldwide pandemic. results of current research demonstrating the mechanisms that lead to inflammation of the nervous system, highlighting the malfunction of the immune system and the most affected cells, such as astrocytes and experts pericytes are cells present in blood capillaries throughout the body, including those that supply nerves
- In addition, COVID-19 has a high affinity for ACE2 (Angiotensin-2 Converting Enzyme), whose function is related to the regulation of blood pressure, therefore, its expression occurs to promote cardiocerebral vascular protection and attenuate the mechanisms of blood pressure atherosclerosis. However, there is high expression of this enzyme in the nasal mucosa, particularly in the ciliated epithelium and cup cells, where viral replication seems to be the greatest, as evidenced by the highest viral titers in the nose
- SARS-CoV-2 infection often causes increased luminal pressure in blood vessels, coagulopathy and prothrombin time, factors that contribute to secondary cerebral hemorrhage. Other studies show the virus can trigger a cytokine storm - an inflammatory response uncontrollable by the immune system - can develop a hyper-inflammatory syndrome, leading to death, and infect macrophages and glial cells that are capable of secreting pro-inflammatory factors
- The relationship of high levels of IFN-1 in the late phase of the disease results in more harmful outcomes due to the activation of the cGASSTING pathway generated by the infection, which is involved in the destruction tissue in response to IFN-1 production. This response can lead to exacerbated inflammation and damage to Central Nervous System (CNS) tissue

Biography

Medical student at the Anhembi Morumbi University. Researcher at the University of Sao Paulo (USP) - Department of Immunology. Research grant from FAPESP for the project "Elimination of RASSF9 expression in B16F0 melanoma cell line by CRISPR/Cas9 technology". Chapter Publisher about transcranial stimulation in neuropsychiatry diseases. Researcher in Institute of Memory, Group studies of Neurosciences ONG - Doctors of the World, Oncogenetics and Oncogenomics, and Infectology. Lastest article accepted for publication “Letter: Importance of Cobalt-60 Dose Rate and Biologically Effective Dose on Local Control for Intracranial Meningiomas Treated with Stereotactic Radiosurgery. NEUROSURGERY, 2022”. She has interests in Genetics, Immunology, Immunotherapy, Precision Medicine, Neurosurgery, Neuroimmunology, Neurology.

VIRTUAL

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The effects of neurotrophin-3 in zebrafish parkinson model

Parkinson's Disease (PD) is a multisystem neurodegenerative disorder characterized by heterogenous clinical features associated with lewy bodies and loss of dopaminergic neurons in the substantia nigra pars compacta. Currently, PD is managed using several disease-modifying agents that aim on managing but unfortunately not to cure the disease. Neurotrophins are diffusible peptides secreted from neurons and glial cells that have gained increasing popularity for their neuroprotective role in parkinson's neurodegenerative diseases treatment. Hence, this study aims we want to look for identify the effects of a relatively recent neurotrophin, neurotrophin-3 in zebrafish PD model. In this study, we induced PD was induced in zebrafish using 1-Methyl-Phenyl-1,2,3,6-Tetrahydropyridine (MPTP, 100µg/g) intraperitoneal injection. Following induction, the zebrafish received recombinant neurotrophin-3 protein (rNT3) via CerebroVentricular Intracranial Microinjection (CVMI). The animals were assessed on day-3, day-5, and day-10 of the post-treatment. The locomotor assessment showed a significant improvement in the exploratory behavior of the fish from the time spent on the top half of the tank and the latency to reach the top ($p<0.05$). The speed and total distance have improved for all treatment groups but did not reach statistical significance. The expression of th1, dat, ntf3, and bdnf gene genes has have also shown significant changes compared to their respective PD models ($p<0.05$). Following that, the chromogenic immunohistochemistry Tyrosine Hydroxylase (TH) staining improved significantly in PD-specific regions in the treatment group compared to the PD model ($p<0.05$). NT3 has been shown to provide benefits in the zebrafish PD model. More studies need to be conducted on the treatment regime and potential drug carrier to transport NT3 across the blood-brain barrier in humans.

Audience Take Away:

- The role of neurotrophin-3 in Parkinson's disease in an in vivo study
- This is the first study conducted in the zebrafish model using this neurotrophic factor. We have established the dosage required for a zebrafish model, and the molecular assessment following treatment
- This study will open more ideas and opportunities for using different types of neuronal growth factors in searching for a cure for Parkinson's disease
- The NT3 has also shown features of anxiolytics in the locomotor assessment, hence may provide benefits in the study for anxiety disorder as well

Biography

Dr. Omar is a Ph.D. candidate at Malaysian National University supervised by Prof. Teoh and Dr. Kumar. The current project is mainly on neurodegenerative disease, Parkinson's disease. Previously, Dr. Omar completed her master's degree in human anatomy at The University of Edinburgh with distinction and completed her undergraduate degree in medicine at the National University of Malaysia. Prior to her Ph.D. project, Dr. Omar was working as a full-time medical lecturer at Universiti Sains Islam Malaysia and has published a workbook in Human Anatomy and several publications pertaining to Anatomy medical education and neuroscience.

VIRTUAL

Nishi M Satish*, Kritika Chopra, Sangeeta Abrol
Department of ophthalmology VMMC and Safdarjung Hospital, New Delhi, India

Lebers hereditary optic neuropathy: The importance of a multidisciplinary approach

Lebers Hereditary Optic Neuropathy (LHON) is a mitochondrial genetic disease that preferentially causes blindness in young males, prevalence between 1 in 8500 and 1 in 50000. It is an inherited neuropathy characterized by bilateral subacute loss of central vision owing to focal degeneration of the retinal ganglion cell layer and optic nerve. Classically associated with the mitochondrial base-pair mutation G11778A; only 50% of males, and 10% of females who harbour a pathogenic mtDNA mutation develop optic neuropathy. This marked incomplete penetrance and gender bias suggests the role of secondary precipitating factors which may modulate the phenotypic expression, contributing to the onset of visual loss.

We report a case of a 14-year-old boy presenting with bilateral painless, progressive diminution of vision, which was noticed eighteen months ago. On examination, the left eye had grade 1 Relative Afferent Pupillary defect. Fundus examination revealed temporal pallor of the optic disc in left eye, while the right eye was normal. Visual Evoked Potential showed a decreased amplitude of P100 in both eyes. A genetic study was planned, which revealed G11778A mutation, thereby confirming our diagnosis of LHON.

This report describes the natural history of LHON. It highlights the basis for differential diagnosis of visual loss in childhood. It presents the findings of recently published studies and the insight they provide on the complex pathophysiology of LHON. It discusses management modalities and emphasizes upon the importance of genetic counselling.

Audience Take Away:

- This case report can help make the clinical diagnosis of Lebers Hereditary Optic Neuropathy
- In a country like India, it is important to understand that not all suspected patients can undergo genetic testing to confirm or rule out this entity, when they present with a plethora of symptoms. To establish the clinical diagnosis of LHON as well as entities like LHON plus, an inquisitive eye is needed to catch the characteristic features of this disease. An interdisciplinary approach with the help of neurologists, paediatricians is also needed to manage the systemic effect this disease entity will have on the patient. Furthermore we stress the importance of the psychiatric counselling this patient will surely need in view of the devastating consequences of the disease. Genetic counselling is also stressed upon when members of the patients family are confirmed to have the mutation as well

Biography

Dr. Nishi Meghna Satish is currently a Post graduate student for the degree of MS Ophthalmology, in Vardhman Mahavir Medical College and Safdarjung Hospital,New Delhi. She has done her MBBS from MVJ Medical College and Research Center Bangalore.

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24-26 OCT

DAY 02

KEYNOTE FORUM

6TH EDITION OF

INTERNATIONAL CONFERENCE ON

NEUROLOGY AND BRAIN DISORDERS

An alternative approach to gait inertia and failed righting reflexes

There exist many theories regarding the basis of gait inertia and instability for people with Parkinson's Disease (PD). This presentation will use a video to demonstrate an alternative explanation for this occurrence together with a very simple mode of intervention, based on the novel use of a walking stick, as an aid to mobility. The hypothesis is based on the gait inertia and instability which occurs as a consequence of the rigidity that accompanies PD and results in a slow response to disturbance of balance which translates to a propensity to falling, when such imbalance occurs, as occurs to all people, including those who do not have PD. The person with PD lacks the capacity to accommodate and compensate for that disturbance of balance and thus is prone to falls and self-harm. Such difficulty with balance, due to failed righting reflexes and the ability to protect against falls, causes the person to experience a fear of moving forward and this can result in inertia of gait and the need for intervention to reinstate a level of confidence. What this video show is that the proper use of a walking stick has the capacity to overcome much of the fear of falling by acting as an adjunct to moving the 'centre of gravity' forward when walking. It acts by providing a different form of intervention which is simple, inexpensive and based on common sense which allows a new level of reassurance which supports the patient's enhanced mobility in a very simple fashion that employs a different way to walk with the aid of a walking stick and as a consequence returns a level of confidence which the patient has lost.

Audience Take Away:

- The audience will gain a new perspective on the possible cause of gait inertia and falls for people with PD
- The video will demonstrate the capacity to compensate for failed righting reflexes
- The presentation explains that which affects people with PD in a different and very simple fashion that all clinicians can instigate within their practices without significant expense
- This presentation has the potential to change the way clinicians treat their patients with PD without cost to them other than patient education

Roy G Beran

Neurology Department, Liverpool Hospital, Liverpool, NSW, Australia

Conjoint Professor, South Western Sydney Clinical School, University of NSW, Sydney, NSW, Australia

Ingham Institute for Medical Research, South Western Sydney Area Health Service, NSW, Australia

Professor, School of medicine, Griffith University, Southport, Qld, Australia

Conjoint Professor, School of Medicine, Western Sydney University, Sydney, NSW, Australia

Biography

Roy G. Beran is trained as a consultant neurologist and accredited sleep physician, in addition to working within legal medicine, military medicine and aviation medicine. His qualifications include: MBBS, MD, FRACP, FRACGP, Grad. Dip. Tertiary Ed., Grad. Dip. Further Ed., FAFPHM, FACLM, FRCP, FAAN, FACBS, B Leg. S, MHL and FFFLM (Hon). He is registered with the Australian Health Practitioner Regulation Agency (AHPRA) as a specialist in Neurology, Public Health and Sleep Medicine and was a Designated Medical Examiner for the Civil Aviation Safety Authority, a medical assessor for Dispute Resolution for the State

Insurance Regulatory Authority and an assessor for the Workers Compensation Commission of New South Wales (NSW). He is a Conjoint Professor of Medicine at the University of NSW; Professor in the School of Medicine at Griffith University, Queensland; and Professor, Chair, Medical Law, Sechenov Moscow 1st State University, Moscow, Russia. He was the inaugural Visiting Professor at the International Research Institute of Health Law Sciences at the Southern Medical University, Guangzhou, Guangdong Province, China. He was also a visiting professor to the Macau University and is co-editor of the textbook, 'Legal Liability in Asia and Australasia', with Prof Raposo from that university. He is: a founding Fellow of the Australasian College of Legal Medicine; a Past President of the College, having stepped down in 2011, while remaining on Council, and was awarded the second ever Honorary Life Fellowship of the College. In 2019, he was appointed as 'Co-Head of Faculty' to convene and co-ordinate the College's training courses throughout Australia. He is the Australian Governor and was the Secretary General and remains a Vice President of the World Association for Medical Law (WAML), having served on the Organizing Committee of numerous World Congresses on Medical Law, presided over the World Congress, in Sydney, in 2004, and successfully bid to host the World Congress, to be held on the Gold Coast, Queensland, in 2022. He was the first Honorary Fellow of the Faculty of Forensic & Legal Medicine of the Royal College of Physicians (London).In Neurology, he is: a Fellow of the Royal Australasian College of Physicians; a Fellow of the Royal College of Physicians, Edinburgh; Corresponding Fellow of the American Academy of Neurologists; and a Member of the Australian and New Zealand Association of Neurologists. He is a Member of the Australasian Sleep Association, serving on various committees thereof. He pioneered the conduct of clinical trials and undertaking research within private practice, having been principal author in publications which included leading academic/tertiary referral institutions. He has published more than 360 papers, book chapters and letters to the editor, presented in excess of 400 papers at national and international meetings and written or edited 17 books, including 'Legal and Forensic Medicine', and is on numerous editorial boards, including being the editor in chief of the international journal, Medicine and Law, for the WAML. His research interests include: Concussion; Epilepsy; Legal Medicine; Stroke; Sleep; Neuroepidemiology; and Medical Education. He was an officer in the Royal Australian Navy Reserve (holding the rank of Commander) and was awarded membership of the general division of the Order of Australia, in 2015.

BOOK OF ABSTRACTS

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POSTERS

6TH EDITION OF

INTERNATIONAL CONFERENCE ON

NEUROLOGY AND BRAIN DISORDERS

VIRTUAL

Ryane Elizabeth Adams

Neurology Department, University of Texas at Houston Health and Science Center / McGovern Medical School Houston, TX USA

A case of myelin oligodendrocyte glycoprotein antibody disease presenting similar to multiple sclerosis

Myelin oligodendrocyte glycoprotein antibody disease (MOGAD) is defined as an autoimmune disease of the CNS associated with a serological antibody against myelin oligodendrocyte glycoprotein (MOG). MOG is found within the CNS: the optic nerves, brain, and spinal cord. Clinically, the disease can resemble multiple sclerosis (MS) and neuromyelitis optica spectrum disorders noted by relapses of optic neuritis and transverse myelitis. We report a patient who was brought to clinic for a second opinion after being diagnosed with multiple sclerosis for many years with remitting and relapsing symptoms before experiencing complete paraplegia of the lower extremities and presenting with new symptoms inconsistent with multiple sclerosis and more concerning for MOGAD.

Case Presentation: A woman in her early forties presented to clinic for a second opinion after being diagnosed with multiple sclerosis (MS) in 2004. Initial symptoms included left sided paresthesia following the birth of her first child. Her initial MRI showed spinal lesions and she began Aubagio for treatment. Her MS was characterized by multiple relapses with good recovery until August 2020. In August 2020 the patient experienced complete paraplegia and lost all sensation of the lower extremities with no since recovery. Her neurological exam was notable for both upper and lower motor neurons signs such as complete absent strength, movement, sensation, reflexes, and hypotonia in the lower extremities, but brisk reflexes bilaterally in the upper extremities. Most recent MRIs showed spinal cord lesions at C1-C3, T8, T12/L1 and heterogeneous patchy abnormal edema of the spinal cord at the level of the inferior brainstem and spinal cord junction suggestive of nonspecific myelopathy. On a follow-up visit, additional symptoms reported included increased fatigue and hair loss, but no urinary/bowel symptoms or incontinence, tremor, swallowing difficulties. Medication was changed from Aubagio to Rituximab and NMO antibody testing is pending.

Discussion: Distinguishing myelin oligodendrocyte glycoprotein antibody disease from multiple sclerosis is critical as the course of disease and treatment vary vastly between the two pathologies. However, the similar, overlapping symptomatology between the two diseases add a level of complexity and can delay proper diagnosis and treatment. Knowledge of the complete clinical spectrum of MOGAD is incomplete, specifically when comparing inflammatory demyelinating diseases such as multiple sclerosis.

Audience Take Away:

- Pathophysiology and presentation of myelin oligodendrocyte glycoprotein antibody disease (MOGAD)
- Differentiate myelin oligodendrocyte glycoprotein antibody disease and other demyelinating diseases, specifically multiple sclerosis
- Broaden the spectrum of knowledge of myelin oligodendrocyte glycoprotein antibody disease to aid in more accurate diagnosis and proper treatment

Biography

Ryane Adams' studied Biology at Xavier University of Louisiana in New Orleans, Louisiana and graduated in 2018. She is currently a 4th year medical student at the University of Texas at Houston Health and Science Center located in the Texas Medical Center. She is applying to Physical Medical and Rehabilitation this application cycle with the hopes of becoming an academic physiatrist with a focus in neurorehabilitation and spasticity.

VIRTUAL

Jacob Saucier*[1,2], Zaynab Beroual[1,2], Caroline Jose[1,3], Kendra Cooling[3], Simon Chartrand[1,2], Mohammad Al-Qadi[1,2], Eméraldine Libert[1,2], Marie-Claire Losier[3], Gabriel Girouard[3], Jalila Jbilou[1,2,4], Ludivine Chamard-Witkowski[1,2,3]

[1]Faculte de Medecine et des Sciences de la Sante, Université de Sherbrooke, Sherbrooke, QC, Canada
[2]Centre de Formation Medicale du Nouveau-Brunswick, Moncton, NB, Canada
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Cognitive inhibition deficit in post COVID-19 syndrome

Background and Objectives: As the COVID-19 pandemic progresses, an increasing amount of research points towards the importance and prevalence of long-term neurocognitive symptoms following infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2).

Design: A PCR test confirmed SARS-CoV-2 infection in our 134 participants at least 12 weeks prior to the interview. Questions focusing on sociodemographic information, past medical history and COVID-19 infection were administered over the phone, followed by standardized neuropsychological tests and health questionnaires screening anxiety, depression, fatigue and autonomy.

Results: This study is the first to observe prefrontal inhibition deficits with the Hayling test. An impairment of cognitive flexibility and inhibition was revealed in 38.8% of participants (n=52), as assessed by an abnormal score on the neuropsychological test. Severity of depression, hospital or intensive care unit (ICU) admission and the duration of hospital or ICU stay predicted an abnormal Hayling score. An abnormal TICS evaluation in 19.4% of participants (n=25) showcased a global impairment of cognition and cognitive efficiency, while 13.4% of participants (n=18) had an abnormal oral Trail Making Test (TMT) result. Executive dysfunction was screened in 56.0% of patients (n=76) as evidenced by an abnormal TMT test or Hayling test. Furthermore, 56.7% of participants (n=77) showed signs of cognitive impairment, receiving an abnormal score on at least one neuropsychological assessment. Regarding general health questionnaires, 55.2% of patients (n=74) had symptoms of psychiatric illness, while 21.6% (n=29) of patients had moderate to severe anxiety or depression. Specifically, GAD-7 questionnaire screened anxiety of mild severity in 20.9% of patients, of moderate severity in 7.5% of participants and severe anxiety in 5.2%. Regarding depression, the PHQ-9 questionnaire revealed mild symptoms of depression in 20.9%, moderate depression in 11.9%, moderately severe depression in 3.7% and severe depression in 3.0% of study participants. Fatigue was screened in 70.9% of patients. (n=95).

Discussion: This study supports the extensive literature on the cognitive and neuropsychiatric sequelae of COVID-19. Hayling's test results suggest long-lasting inhibition deficits, validating and expanding on past Stroop test data. Devoid of correlation between the duration elapsed since the initial infection and the interview, this study suggests that persistent cognitive impairments may not improve with time. Furthermore, statistically significant correlations between the severity of depression and inhibition deficits after COVID-19 infection shed light on similar pathophysiological mechanism, and the interesting interplay between depression and cognition in post COVID-19 syndrome. The orbitofrontal cortex plays a crucial role in inhibition functions, in addition to social and emotional regulation. Thus, these frontal inhibition deficits lend support to the hypothesis that SARS-CoV-2 can invade the CNS from the olfactory tract, due to the anatomical contiguity of the cribriform plate and the orbitofrontal cortex.

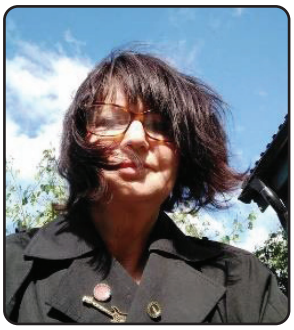
Conclusion: It is becoming evident that there is an ever-increasing need for physical and cognitive rehabilitation in patients who have had COVID-19. Future research should assess inhibition deficits in a longitudinal matter to assess the progression of these impairments.

Audience Take Away:

- Due to the significant prevalence and social burden of post COVID-19 syndrome cognitive sequelae, it is important for clinicians to be aware of newly discovered deficits. The audience will learn about the prevalence of inhibition deficits in post COVID-19 syndrome patients, how to recognize such deficits and their consequences
- The audience will learn about mechanisms underlying inhibition deficits in COVID-19 and its close relation with depression. An intriguing interplay between these two prevalent COVID-19 sequelae leads to a vicious cycle, emphasizing the need for longitudinal care and therapy in post COVID-19 syndrome patients
- By illustrating the important relation between the severity of COVID-19 infection and subsequent cognitive impairment, the audience will gain insights on the how greater infection severity can translate to neuronal injury, leading to a significantly higher frequency and severity of cognitive impairment. In the clinical setting, this will allow clinicians to anticipate persistent cognitive deficits in a population, allowing them to address their longitudinal need for care and rehabilitation

Biography

Jacob Saucier is a third-year medical student at the University of Sherbrooke, in Canada. His great interest in neurosciences has been reflected by his participation in protocol redaction, literature review, data collection and manuscript redaction of numerous neurology projects at the George L. Dumont University Hospital Centre. Being the first author of articles spanning from NeuroCOVID to Spinocerebellar Ataxias and having presented his research at the national and international level from Quebec to France, it is evident that Jacob Saucier is very passionate about neurology and contributing to its exciting and ever-expanding field.



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Gait and balance among people with de novo vs. mild to moderate parkinson's disease

We have investigated how a de novo cohort of people with Parkinson's Disease (PD) differs in terms of balance and gait function as well as frequency of near falls and falls in comparison to a cohort with mild to moderate PD that has been medicated for several years. Preliminary results (Table 1) showed that people with de novo PD have balance and gait impairments and that some have also experienced near falls and falls. On the contrary, the cohort with more advanced disease has significantly fewer motor symptoms, as shown in the UPDRS motor score, compared to those newly diagnosed. This is probably related to successful symptomatic medication in the more advanced cohort. Despite less motor symptoms, those with a more advanced disease performed significantly worse on the balance-demanding tests (i.e., Timed Up and Go, Tandem Gait) and reported more near falls and falls. These results are consistent with previous studies reporting that dopaminergic medication improves motor symptoms, but has less effects on mobility and balance in PD. This indicates a need for balance-promoting efforts from an early stage of the disease.

Table. Comparision of people with de novo and mild to moderate Parkinson's Disease (PD)

	De novo PD N=48	Mild to moderate PD N=58	P- value
Female gender	21 (43)	32 (55)	0.104
Age (years)	67 (11.5; 42–83)	69 (8.9, 46–84)	0.216
PD duration (years)	NA	6.7 (3.68, 3.6–20,5)	NA
Daily total levodopa equivalent (LDE) dose (mg)	NA	500 (400–675, 150–1580)	NA
Motor symptoms (UPDRS III, higher= worse)	18 (8–53; 14–25)	13.5 (9–20; 1–40)	0.003
Retropulsion (NRT, higher=worse)	0 (0–0; 0.2)	0 (0–1; 0–3)	0.283
Comfortable Gait speed, m/s (10MWT, higher=better)	1.1 (0.28; 0.6–1.6)	1.1 (0.31; 0.4–1.7)	0.588
Mobility, s (TUG, higher=worse)	10.6 (3.85; 7.0–28.9)	12.4 (8.57; 7.2–61.5)	0.023
Dynamic balance (TG, higher=worse)	0 (0–2; 0–3)	2 (0–3; 0–3)	0.027
Number of individuals with near falls	2 (4)	18 (31)	< 0.001
Number of individuals with falls	3 (6)	30 (52)	< 0.001

Values are mean (SD; min–max), n (%), or median (q1–q3; min–max).
Mann–Whitney U-test and Chi 2 test were used.
NA, Not Applicable; NRT, Nutt Retropulsion Test; 10MWT, 10-Meter Walk Test; m/s, meters per second; TG, tandem gait; TUG, Timed Up and Go test, s, seconds; UPDRS III, part III (motor score) of the Unified Parkinson's Disease Rating Scale.

Audience Take Away:

- As we previously mentioned, our results are consistent with previous studies reporting that medication has little effect on balance symptoms. The anti-Parkinson medication improves some of the motor symptoms of PD but has less effects on mobility and dynamic balance. This indicates a need for balance assessment already in an early stage of the disease i.e., at the time for diagnosis. Balance investigation should target different aspects of balance (reactive, dynamic, static) as well as mobility. Furthermore, asking about near falls or falls should also be included already at the time for diagnosis. Then, all these parameters should be monitored over time
- What are potentials reasons for progress of balance impairment over time? What happens in the body
- How to investigate changes in balance performance over time
- What can be done to prevent progression of balance impairment

Biography

Certificated Physiotherapist. approved by physiotherapists as a specialist in neurology 2013. Employed as a physiotherapist at Skåne University Hospital, Malmö, Sweden since 1990. Worked mainly with neurological rehabilitation. Since 2002 active member of Movement disorders team at Skåne University Hospital. Participated in the National Board of Health and Welfare's work with National Guidelines for Parkinson's / Multiple Sclerosis as an external expert in the priority group (2015–2016 and 2022). Received PhD degree in 2017 at Lund University (LU), Sweden. Since then, combines clinical work and patient-centered clinical research as postdoctoral follow at Clinical Memory Research, LU, and strategic research area MultiPark.

VIRTUAL

Gabriela Dumitrita Stanciu*[1], Bogdan Ionel Tamba[1,2]
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Preclinical in vivo study on selective cannabinoid receptor type 2 and donepezil combined therapy in alzheimer's disease

As one of the major healthcare challenges worldwide, Alzheimer's Disease (AD) is a detrimental brain disorder of a multifactorial nature, with an etiopathogenesis still not completely clear, influenced by epigenetic and genetic variants combined with environmental and lifestyle factors. With over 50 million people afflicted by AD globally, and total worldwide payments of caring for these patients estimated at $1 trillion in 2020, Acetylcholinesterase (AChE) inhibitors remain the mainstay treatment option, providing only symptomatic relief without slowing the disease progression. The cholinesterase inhibitors that interact simultaneously with AChE (catalytic and peripheral sites) and amyloid-beta (Aβ) plaque deposition coupled with added properties such as antioxidant action, neuroprotective activity or endocannabinoidergic system display the potential of ameliorating the cognitive deficit in AD by restoring cholinergic activities. The endocannabinoid system is composed of at least two well-described Cannabinoid receptor 1 (CB1) and receptor 2 (CB2). CB1 receptors are widely expressed in the central nervous system, where they regulate the main functions of the brain. Recent studies have suggested that CB1 receptor-specific agonists have potential therapeutic properties in AD at low non-psychotropic doses. However, major attention in AD has been paid to specific CB2 receptor agonists due to their lack of psychoactive properties, even though CB2 receptors are mainly expressed in the immune system, with relatively low expression in neurons. Interestingly, it has been found that CB2 receptors are selectively overexpressed in cells associated with Aβ enriched neuritic plaques in AD samples from postmortem human brains. In light of all these data, exogenous and endogenous cannabinoids represent an attractive and promising target for the treatment of AD. So far, no results have been published to determine the effects of donepezil and CB2 agonists' co-administration on AD-relevant behaviours and brain pathology.

The aim of this study was to evaluate the effects of long-term donepezil- cannabinoids with CB2 selectivity co-treatment in AβPP/PS1 mice, a transgenic model of AD that mimics the progressive cognitive deficiency and neurodegenerative process, in pre-symptomatic and early stage of the symptomatic phase of the disease.

In vivo studies supported by cognitive performance evaluation, multimodal imaging, histology and immunohistochemistry analysis were used to meet the objectives of this study, investigate the preclinical efficacy of long-term co-treatment associations, evaluate the impact of therapy on cerebral metabolism, identify the acetylcholine-related changes and amyloid-β quantification in an animal model.

Translating the selective CB2 agonists into the clinic is not an easy challenge, but identifying innovative approaches for treating millions of AD people holds the promise of improving their daily life and CB2 agents might just prove to be effective, due to their modulation of the endocannabinoid system, as well as their effect on neuroinflammation, Aβ clearance, cell viability in the presence of Aβ, and glucose up take in brain.

Audience Take Away:

- Identify important limitations of animal models in the investigation of cannabinoid effects in the Alzheimer's disease, and focus future research in this area on specific, unanswered questions regarding the complexities of endocannabinoid system and its main receptors (CB1 and CB2)
- The role of age in determining the long-term effect of cannabinoid on cognition, highlighting possible detrimental consequences during brain development and beneficial outcomes in old age
- A high degree of experimental control can be achieved by precisely controlling the animal's life experiences and history of drug exposure. This allows clear inferences to be made concerning the causality of effects observed in the experiment
- The brain mechanisms that underlie addictive behaviour, procedures that can be conducted in humans are severely limited compared with the manipulations and measures that can be performed in animals

Biography

Dr. Stanciu is a researcher who has continuously improved her knowledge and expertise, **translating basic research into neurological practice.** Holding a Medical Doctor degree (USAMV, Iasi and Ecole Nationale Veterinaire d'Alfort Paris) and a PhD degree in Neurophysiology (USAMV and Royal Veterinary College University of London). Throughout her scientific career demonstrated abilities and scientific interests in preclinical neurophysiology and neurodegenerative diseases and especially in Alzheimer's disease. The continued interest in research on future therapies for the prevention and treatment of AD as well as Dr. Stanciu's extensive experience as member in larger projects, lead to her being awarded with 2 grants the publication of over 50 articles research in ISI journals.

VIRTUAL

Shaymaa Al Umran, Fatimah Al Shakhs, Basmah Saeed Al Zahrani, Ali Al-Ahmad, Mariam Al-Umran*, Reem Al Marshad, Basit Ali Syed

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Intracranial extramedullary hematopoiesis as "Ghost Tumor (Meningioma)": First case report in sickle cell disease and review of literature

Extramedullay Hematopoiesis (EMH) is a common compensatory phenomenon to chronic hemolytic anemias. Commonly occurs in the liver, spleen, kidney, lymph nodes and also been reported in skin, adrenal gland, thymus, ovary, paraspinal regions of thorax, pelvic region and the process can often manifest as a mass or mimicking neoplasm. An Intracranial EMH is an extremely rare occurrence and has been more commonly reported in patients with Thalassemia, Myelofibrosis and we did not come across a report in Sickle Cell disease. We are reporting a 26 year-old male with sickle cell disease, who present with generalized seizure and was diagnosed elsewhere to have a left sphenoid wing meningioma and was offered surgery but he refuse at that time. One year later, patient presented to our ER with generalized seizure and wants to go for surgery. His repeat MRI Brain revealed no lesion suggesting complete spontaneous resolution previously noted lesion (tumor). We present this case and review the related literature.

Audience Take Away:

- Don't rush to do a final diagnosis without full work up and an opinion of your senior colleagues
- Rare and unusual cases needs to be investigated individually and in multidisciplinary panel
- If its rare case, then reporting it is a good idea

Biography

Mariam Alumran, medical student at Alfaisal university, Riyadh, Saudi Arabia. She is working on clinical researches in different medical specialties.

VIRTUAL

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Nicotine's effect on neurons and on the brain

Approximately 50 million people are addicted to nicotine just in America, worldwide these numbers reach 1.1 billion with 6 million people dying each year from forms of smoking. Even though nicotine is generally known as the "bad guy", it is important to recognize the bad and good types of it. Nicotine is a naturally occurring alkaloid that can be found in a wide variety of plants such as tomato, potato, eggplant (aubergine), cauliflower, tea, green pepper, and coca plant's leaves; however, it is best known as the highly addictive substance found in tobacco. To put it in simple words, nicotine is a stimulant drug that speeds up the neurons traveling between the body and the brain. Nicotine mostly affects the prefrontal cortex in the brain which is responsible for emotions and impulse control. Subsequently, if nicotine is used in teen years, it can obstruct aspects of that development, resulting in long- term brain damage. It can also lead to an increase in blood pressure, heart rate, blood flow to the heart, artery constriction (vessels that carry blood) as well as artery wall hardening, which can lead to a heart attack. Nicotine affects behavior: enhances mood and attention at first, reduces anger and tension, relaxes muscles, and decreases appetite. As mentioned above, nicotine is mostly known for causing addiction: Research shows that smokers can get addicted to nicotine in just a few days. It imitates a number of neurotransmitters that transmit messages in the brain, nicotine's structure is most similar to that of the neurotransmitter acetylcholine, therefore signaling in the brain increases when nicotine enters. The brain begins to reduce the number of acetylcholine receptors overtime to compensate for the increasing signaling activity. Nicotine tolerance develops, as a result, necessitating the usage of more nicotine in the future. Nicotine also activates the brain's pleasure regions, simulating dopamine, causing your brain to equate nicotine usage with feeling good, nevertheless, nicotine's enjoyable sensations don't endure very long. To receive the next thrill, a person needs to keep inhaling nicotine. Chronic nicotine stimulation desensitizes GABAergic neurons, causing them to lose their inhibitory impact on dopamine; this, in turn, feeds the addiction by causing cravings. The chemistry of addiction, nicotine structure and nicotine's effects on people will be further discussed in the research.

Keywords: Nicotine, Addiction, Acetylcholine, Stimulant, Dopamine, Neurons, Neurotransmitters, Tobacco, Brain, Behavior, GABAergic

Audience Take Away:

- What is nicotine? What is the chemical structure of nicotine
- Chemistry of addiction to nicotine
- Nicotine's effects on the brain, neurons, and behavior
- Recognizing good and bad types of nicotine
- Learning where nicotine is found

Biography

Begüm Bulgurluoğlu was born in 2005 in Istanbul, Turkey. She will graduate from Hisar High School in 2023. Her papers "Effects of Childhood Abuse On Adulthood" and "Dear Diary" were published in Columbia University Books: Steam Punks and Origin Stories; "Impacts of Sleep On Memory and Learning" and "Benefits of Artificial Intelligence in Medicine" on Hisar Science Newsletter; few more articles on the School newspaper "Hisarın Sessi".

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Sex differences in clinical and functional outcomes of parkinson's disease: Single center experience in republica dominicana

Background: Parkinson's disease (PD) is usually more prevalent in men than in women, which has been explained in terms of environmental, hormonal and genetic influences, but little has been reported in terms of clinical characteristics among sex.

Objective: To analyze motor, non-motor, neuro-psychiatric and functional characteristics on PD according to sex.

Methods: A single center retrospective dataset was analyzed with treated PD cases in a tertiary hospital in Republica Dominicana. Demographic, clinical and functional outcomes were recorded during the follow-up period. Descriptive statistics were used to compare differences among sex.

Results: A total of 83 cases (41 female [median age 54, IQR 50-60 years] vs. 42 male [median age 54, IQR 49-52 years] were included, with a mean follow-up of 7.6 ±4.6 years for the entire population. No significant differences were found in the main comorbid conditions, previous exposition to pesticides or family history of PD between male and females cases. When comparing female cases vs. male cases, motor symptoms (resting tremor [87.5% vs. 97.6%] , Rigidity [90% vs. 88.1%], and bradykinesia [85% vs. 92.9%]) presented no differences among groups. Non-motor functional manifestations (constipation, bowel incontinence, weight loss, nocturia, hyposmia), presented a higher non-significant tendency in male cases. No differences were found in neuro-psychiatric manifestations. When analyzing functional outcomes with the Hoehn & Yahr scale, 50% of the 8 cases with H&Y=4 presented with 100% frequency of gait disturbance and dysphagia, with no differences in the rest of categories (p=0.79). In terms of therapeutic options, Dopaminergic agonists (52.2% vs. 73.8%, p=0.04) and Anti-depressive agents (15.8% vs. 2.8%, p=0.05) were more common prescribed in female cases. Finally, Only 5 early onset cases (<50 years) were detected, with no significant differences (p=0.62).

Conclusion: No significant differences were found in motor, non-motor and functional characteristics in PD cases when compared between sex.

Biography

Dr. Marcia Castillo performed her internal medicine and neurology residency in Santo Domingo, Republica Dominica, with a subsequent Fellowship in Parkinson's Disease and Movement Disorders. She has been performing her clinical practice with emphasis in clinical and epidemiology of PD in República Dominicana, and currently she holds a position at the Dominican Republic PD Foundation.

VIRTUAL

Zhi-Hao Liu*, Zhong-Yuan Yu, Chen-Yang He, Hui-Yun Li, Ying-Ying Shen, Xian-Le Bu, Yan-Jiang Wang

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The impact of APOE4 genotype on the aβ phagocytosis function of blood monocytes

Background: ApoE4 is the strongest genetic risk factor for sporadic Alzheimer's Disease (AD). Previous studies have shown that innate immune dysfunction induced by ApoE4 plays an important role in brain Aβ deposition and AD. GWAS studies showed that mutations in genes related to the phagocytosis function of monocytes significantly increased the risk of AD. We previously found that decreased Aβ uptake ability of blood monocytes in AD patients was involved in the occurrence and development of AD, but the mechanism was unclear.

Objective: To analyze the correlation between ApoE4 genotype and Aβ phagocytosis function of monocyte, which may give a new insight into the mechanism of monocyte Aβ uptake dysfunction in AD

Methods: Blood monocytes of ApoE4 gene carriers (N=30) and non-carriers (N=32) from the cognitively normal population and blood monocytes of ApoE4/4 transgenic mice (N=3) and ApoE3/3 transgenic mice (N=3) were separated by ficoll density gradient centrifugation. The uptake amount of Aβ42 was detected by flow cytometry after co-culture of monocytes with Aβ42-FITC.

Results: Compared with none-ApoE4 gene carriersβAβ42 uptake by total monocytes (CD14dim&CD14+) (p=0.0117), classical monocytes (CD14+CD16-) (p=0.0083), intermediate monocytes (CD14+CD16 +) (p=0.0457) and non-classical monocytes (CD14dimCD16+) (p=0.0230) were significantly decreased in ApoE4 gene carriers. Univariate analysis showed that the Aβ42 phagocytosis function of three subtypes of monocytes is that CD14+CD16+> CD14dimCD16+ > CD14+CD16- (P < 0.0001). In ApoE4/4 transgenic mice, the Aβ42 uptake ability of monocytes (CD115+CD11b+) was significantly decreased compared with that in ApoE3/3 transgenic mice (P=0.0234).

Conclusion: Aβ uptake ability of monocytes in cognitively normal subjects and mice carrying the ApoE4 gene is decreased than that without the ApoE4 gene, which indicates that the ApoE4 gene may be an important cause of monocyte Aβ phagocytosis dysfunction. We speculate that Aβ uptake dysfunction of ApoE4 genotype monocyte may lead to excessive Aβ deposition in the brain and participate in the occurrence and development of AD, which needs to be further investigated in the future.

Audience Take Away:

- Potential of blood monocyte as therapeutic targets for Alzheimer's disease
- Effect of Apoe4 on the phagocytic function of blood monocytes

Biography

Liu studied at the Wenzhou Medical University, Zhejiang, China, and graduated as BD in 2020. He then joined the research group of Prof. Yan-Jiang Wang at the Department of Neurology and Centre for Clinical Neuroscience, Daping Hospital, Third Military Medical University, Chongqing, China. Liu is currently studying for a master's degree in neurology at Wenzhou Medical University. He has published 2 research articles in SCI(E) journals.

Khadga Raj Aran*, Shamsher Singh

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Neuroprotective effect of L- Theanine against tramadol induced parkinson's like symptoms in experimental rats

Introduction: Parkinson's Disease (PD) is a chronic neurodegenerative disorder triggered by degeneration of dopaminergic neurons in Substantia Nigra pars compacta (SNpc). Low antioxidant level, mitochondrial failure and neuroinflammation are the major pathological mechanisms.

Material and Methods: Experimental Animals: Wistar Rats (180-200 g).

Drugs and Chemicals: Tramadol, L- theanine, Naloxone and Sinemet.

Experimental procedure: The animals were divided into 7 groups. Group 1 served as Normal control.

Group 2 received Tramadol (50 mg/kg, i.p.) daily for 28 days. Group 3, 4 and 5 received L-theanine (25, 50 and 100 mg/kg; p.o.) from 14 to 28 day prior to the tramadol administration. Group 6 received Sinemet [levodopa and carbidopa] (36mg/kg; p.o.) from day 14 to day 28 prior to the tramadol administration. Group 7 received naloxone (0.4mg/kg; i.p.) from day 14 to day 28 prior to the tramadol administration. Behavioral observations were done on 1, 7, 14, 21 and 28 day after tramadol treatment. On 29 day, animals were sacrificed and striatum was isolated for biochemical, neuroinflammation, histopathological and neurotransmitters analysis.

Results: Administration of tramadol (50 mg/kg, i.p.) for 28 days in rats produces impaired motor functions and locomotor activity as evidenced by rotarod, open field, narrow beam walk and grip strength performance. In addition, there was increased oxidative stress (MDA, nitrite) and neuroinflammatory markers (TNF-α, IL-1β and IL-17) and decreased levels of catecholamines, GABA and glutamate. The treatment drug L-theanine at dose (25, 50, 100 mg/kg) significantly and dose- dependently improved alterations in motor impairments and locomotor activity, attenuated oxidative stress, neuroinflammatory markers and restored catecholamines, GABA and glutamate level in striatum.

Conclusion: Chronic tramadol administration produces impaired motor functions, increased oxidative stress, neuroinflammation and altered neurotransmitters level was significantly ameliorated by L-theanine, through antioxidant, anti-inflammatory and neuroprotective mechanisms.

Biography

Mr. khadga Raj Aran is presently working as Ph.D. Research Scholar, Department of Pharmacology, I.S.F. College of Pharmacy, Moga. He did his M Pharm from I.S.F, Moga, Punjab, India and currently pursuing PH.D from I.S.F. He is contributing dedicatedly towards Pharmaceutical Education and Research. Mr. Raj is a prolific writer with more than 20 Publications in various reputed peer-reviewed journals to his credit. Besides, this he has contribution of 8 National book chapters related to Human Anatomy and Physiology and 1 book related to clinical pharmacy. He has guided 5 students in UG projects in the area of neurological disorders.

VIRTUAL

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Leukocyte count and neutrophil-to-lymphocyte ratio as simple hematologic predictors of stroke severity and functional outcome in acute ischemic stroke patients

Background: It has long been recognized that inflammation plays a critical role in the pathogenesis of ischemic stroke. However, whether leukocyte count and neutrophil-to-lymphocyte ratio are related to stroke severity and functional outcome is uncertain.

Objective: This clinical study aimed to evaluate the association of leukocyte count and neutrophil-to-lymphocyte ratio with stroke severity and functional outcome in acute ischemic stroke patients.

Methods: This hospital-based, retrospective observational study included 112 subjects with acute ischemic stroke. All subjects had their demographic, clinical, and laboratory data obtained. The leukocyte count and neutrophil-to-lymphocyte ratio were evaluated to stroke severity on admission and 3-month functional outcome. The severity of stroke at admission was measured using the National Institutes of Health Stroke Scale (NIHSS), whereas the Barthel Index was used to measure 3-month functional outcome (BI). We conducted a regression analysis, adjusting for any confounding variables.

Results: Higher leukocyte count was significantly associated with increased risk of stroke severity (odds ratio [or] 1.391, 95% confidence intervals [CI], 1.121-1.725, p: 0.003) and unfavorable functional outcome (OR 1.434, 95% CI, 1.068-1.925, p: 0.017). Higher neutrophil-to-lymphocyte ratio was not significantly associated with increased risk of stroke severity (OR 1.181, 95% CI, 0.947-1.474, p: 0.140) and unfavorable functional outcome (OR 1.246, 95% CI, 0.905-1.716, p: 0.177).

Conclusion: Our study indicates that leukocyte count is an independent predictor of stroke severity on admission and unfavorable functional outcome.

Keywords: Ischemic stroke; Inflammation; Leukocyte count; Neutrophil-to-lymphocyte ratio; Prognosis.

Audience Take Away:

- Prognosis assessment of acute ischemic stroke patients sooner and accurately may minimize the consequences of ischemic stroke
- Our study indicates that leukocyte count is an independent predictor of stroke severity on admission and unfavorable functional outcome
- The findings of this study provide possibilities for future study into the potential utility of anti-inflammatory therapy for patients with acute ischemic stroke

Biography

Vanessa Veronica,B.Med studied medicine at Duta Wacana Christian University, School of Medicine Yogyakarta. She obtained his bachelor of medicine degree in 2021 as the best graduate and is currently undergoing a clinical clerkship and will obtain a medical doctor degree in 2023. She has been actively conducting research in the field of neurology since 2019 and has published 9 studies in international journals.

BOOK OF ABSTRACTS

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NEUROLOGY AND BRAIN DISORDERS

Keith Stenning
Informatics, University of Edinburgh, Scotland

In two minds? Because we need two logics

This is a cautionary tale about disciplinary allergies: a sorry tale from the psychology of reason-ing literature rejecting logic. Understanding a very little logic can be a great boost to empirical investiga-tions. Our goal here is to interest related disciplines in a major shift for empirical work on classical logical reasoning. Logically `naive' participants have been convicted of not understanding classical logic when in truth the Ex-perimenters have not understood how their instructions are interpreted. When put in an unambiguously suit-able context, participants suddenly become quite good at classical logical reasoning, even using some of its horrors to construct their own example countermodels. These properties are routinely used to dismiss any relevance of logic to the psychology of human reasoning. As a control, instructed as the literature does, our participants are just like those from the literature. Worse still, there are already published examples of reasoning in this `right context' (of dispute) which show `classical reasoning' but are not recognised as such. For example, the field simultaneously asserts that partic-ipants are rather good at `simple syllogistic counterexample reasoning', without acknowledging this just is a semantic presentation of classical logic. Lacking a logical map to guide them, they do not recognise when participants' logic changes, or doesn't. There are in fact two contrasting logics (`narrative' and classical). With a good map, the data shows that supposedly `logically naive' participants in constructing their coun-terexamples actually exploit the very features of classical logic that would be absurd in the participants' un-derstanding of the conventional tasks. This upending of the accepted facts points to a range of little explored empirical questions. Both these logics (narrative and classical) are probably largely implicit in these participants: they demonstrably come up with the right output, but they do not have much explicit access to how they reason (Stenning, K., & van Lam-balgen, M. 2022). For narrative reasoning a nonmonotonic logic has been shown to explain the crucial semantic memory re-trieval problem through a neural symbolic network that can be the basis of the huge size of semantic memo-ry, its rapidity of use, sensitivity to exceptions, and the outlines of `reasoning by retrieval' (Stenning, K., & van Lambalgen, M. 2008), and also how such reasoning can be implicit. The work on implicitness shows that illiterate participants can in fact engage this logic with far greater success than they have been credited with. When it comes to disorders, we have presented evidence that mildly autistic participants' discourse process-ing of narrative bears the hallmarks expected from their brain's deficiencies in processing negation (Sten-ning, K., & van Lambalgen, M. 2007; Pijnacker, J. et al. 2009; Pijnacker, J. et al. 2010). ADHD participants show different abnormalities, explicable by their different problems with executive func-tion (van Lambalgen, M., van Kruistum, C., & Parigger, E. 2008). These are `brain symptoms' of rea-soning with narrative logic. For classical logic, the findings presented here are new, relatively undeveloped, and open up widespread em-pirical questions. What implicit knowledge do participants access in what contexts to achieve this classical reasoning? How do those contexts combine with their mental machinery to produce this compliance with the logics? We would like to engage this audience with how classical logical reasoning is exhibited in mind and brain disorders. And don't forget how this reanalysis changes our view of how naive logicians' abilities are transformed by learning/teaching. Instead of bizarre new languages from outer space, logics become regu-larisations. And makings explicit, of what is already known implicitly. References: Pijnacker, J., Geurts, B., van Lambalgen, M., Buitelaar, J., &

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Audience Take Away:

- What is important about logic for psychology and the brain is not firstly the intri-cate technical details, but the broad aims of the discipline: to provide a qualitative mathematics' of reasoning, especially of contrasting goals of reasoning.
- Logics are highly abstract, but they can define specific distinguishing features of contrasting discourses. Narrative, and classical dispute, contrast in their top-level goals: producing new interpretations vs. disputing whether particular proposed in-terpretations are coherent.
- Mental disorders can be traced to features of the implementations of these logics: differences in processing exceptions in autism; achieving continuity in ADHD •Naive classical logical reasoning is about dispute between parties, as opposed to narrative's goal of cooperative communication.
- This broadens the evidence base: qualitative differences in function provide strong evidence of underlying brain implementations.
- This audience knows the most plausible candidates for study better than we do

Biography

Dr.Stenning studied Philosophy and Psychology at Oxford University1966–9. And then a PhD at Rockefeller University, New York in George Miller's group (1975), followed by a PostDoc. After a spell at the Universi-ty of Liverpool, as a lecturer in Psychology, he moved to Edinburgh University at the Centre for Cognitive Science. He was a founding Director of the ESRC funded Human Communication Research Centre from 1990—2000. Two books: Seeing Reason:: Language and Image in Learning to Think, and Human Reason-ing and Cognitive Science (with Michiel van Lambalgen).

BOOK OF ABSTRACTS

INBC 2022

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24-26 OCT

DAY 03

KEYNOTE FORUM

6TH EDITION OF

INTERNATIONAL CONFERENCE ON

NEUROLOGY AND BRAIN DISORDERS

GABA-A receptor modulating steroids impair memory and promotes dementia but can be antagonized with GABA-A receptor modulating steroid antagonists

Gamma-Amino Butyric Acid (GABA) is the main inhibitory neurotransmitter in the brain and GABA-ergic transmission is shown to be of importance for regulation of mood, memory, and food intake. The progesterone metabolite allopregnanolone (Allo) is a positive GABAA receptor modulating steroid with potent effects. In humans, disorders like Premenstrual Dysphoric Disorder (PMDD), hepatic encephalopathy and polycystic ovarian syndrome are associated with elevated Allo levels and increased negative mood, disturbed memory, and increased food intake in some individuals. This is surprising as Allo shares many properties with benzodiazepines and is mainly considered to be anxiolytic and anti-depressant. However, it is well established that in certain individuals GABAA receptor active compounds could have paradoxical effects and thus be anxiogenic in low physiological plasma concentrations while anxiolytic at high levels. In Alzheimer transgenic mice continuous allo in low stress concentrations deteriorated the dementia progress. We have demonstrated that isoallopregnanolone (Isoallo), the 3β-OH sibling of Allo, functions as a GABAA receptor Modulating Steroid Antagonist (GAMSA), but without any effects by its own on the GABAA receptors. The antagonistic effect is noted in most GABAA subtypes investigated so far. *In vivo*, Isoallo inhibits Allo-induced anesthesia in rats, as well as sedation or saccadic eye velocity in humans. Isoallo has been studied in women with PMDD. In two phase II studies, Isoallo (Sepranolone) injections significantly ameliorated negative mood in women with PMDD compared with placebo. One GAMSA, UC1011, could inhibit Allo induced memory disturbances in rats. A GAMSA for oral administration have also been developed, GR3027, has been shown to restore learning and motor coordination in rats with hepatic encephalopathy. In human's, vigilance, cognition, and pathological EEG was improved in patients with hepatic encephalopathy when treated with GR3027.

Torbjorn Backstrom, MD. PhD

Department of Clinical Sciences, Umea University, Umea, Sweden

Biography

Torbjorn Backstrom, MD and PhD, senior professor at the department of Clinical Science, Obstetrics and Gynecology, Umea University as well as head of the Umea Neurosteroid Research Center, started Umecrine Mood together with the Karolinska Institute innovation system in 2006 and the company became Asarina Pharma in 2015. Professor Backstrom's main focus of research since 1972 is the effect of sex and stress hormones on the brain and in particular on the GABA-system, and conditions induced by these hormones. He has published more than 400 scientific publications within this area and he is a frequently invited key-note speaker at scientific meetings around the world. In 1989 professor Backstrom served several years on the scientific advisory board of the pharmaceutical company, CoCensys in the US, a company based on his own and the research of professor Kelvin Gee from University of California. Several active compounds were developed and one of these is currently in phase III of clinical development. CoCensys, was listed on the NASDAQ stock market until 1994, when it was sold to a larger pharmaceutical company.

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24-26 OCT

DAY 03

SPEAKERS

6TH EDITION OF

INTERNATIONAL CONFERENCE ON

NEUROLOGY AND BRAIN DISORDERS

Mohammad Abu-Abaa
Capital Health Regional Medical Center, Internal Medicine Residency Program, Trenton, NJ, USA

Seizure as the main manifestation of non alcoholic wernicke's encephalopathy but without cortical involvement

This is a case report of a 45 years old female patient with a past medical history of depression and poor oral intake presented for a single episode of unwitnessed seizure and 3 days of unsteady gait and vertigo. Then she had two episodes of seizure focal and then generalized tonic. Physical exam was remarkable for lethargy, bilateral gaze induced horizontal nystagmus with rotational component and change in direction. MRI brain with contrast showed non enhancing bilateral symmetrical FLAIR hyperintensities in medial thalami and tectum. Vitamin B1 level was found to be low. Lumbar puncture was unyielding. She was loaded with high dose thiamine replacement. After a few days, a neurological exam revealed improvement with unilateral nystagmus with less lethargy. Valproate that was started initially was eventually discontinued during follow-up after resolution of neurological deficits. Interestingly, a baseline echocardiography showed heart failure with reduced ejection fraction at 40% with clinical euvolemia. It was believed to be secondary to beri beri.

This case serves to remind us that seizure can be the main manifestation of Wernicke's Encephalopathy (WE) even without cortical involvement. It also shows that early thiamine replacement can result in significant neurological improvement. We believe this is a good case to report as seizure as initially presentation of WE was reported only in 4 cases in English literature. In this case report, we aim to review what the English literature has to answer the questions of differences between alcoholic and non alcoholic WE, risk factors of non alcoholic WE, typical and atypical MRI findings, and prevalence of seizure and cortical lesion in WE.

Audience Take Away:

- Seizure is a rare but possible initial presentation of WE
- Differences between alcoholic and non alcoholic WE
- Risk factors for non alcoholic WE
- Typical and atypical MRI findings in WE

Biography

Dr. Abu-Abaa graduated from the University Of Basrah College Of Medicine in Iraq in the top 5% of his class. Following graduation, he completed a Transitional Year of training followed by two years of Internal Medicine and six months of neurology training experiences. Dr Abu-Abaa also volunteered in underserved areas with the Iraqi Ministry of Health. He is currently PGY2 at Capital Health Regional Medical Center Internal Medicine residency program.

VIRTUAL

Geetanjali Rathore, MD
Department of Paediatrics and Neurosciences, University of Nebraska Medical Centre, Omaha, Nebraska

Pediatric autoimmune encephalitis - Finding a consensus on diagnosis and management

Pediatric AutoImmune Encephalitis (AIE) is emerging to be more common than had previously been thought in children. Even though the underlying pathophysiology is similar, the presentation, antibody types, treatment and outcomes in children are very different from adult autoimmune encephalitis. He would like to give an overview of autoimmune encephalitis to begin with then discuss the presentation of this condition in children. He will present the literature on how to diagnose AIE in children, what are the common antibodies to look for, discuss international consensus on management of these paediatric patients and what tumour screening they require. Towards the end, He would like to share experience and provide an opportunity for audience to ask questions and discuss pediatric autoimmune encephalitis.

Audience Take Away:

- Understand the research behind identifying pathophysiology of autoimmune encephalitis
- Understand the presentation and classification of Pediatric autoimmune encephalitis
- Learning how to manage and diagnose pediatric autoimmune encephalitis
- Review current consensus on diagnosing and treating pediatric autoimmune encephalitis
- Share my research experience and data of 10 years

Biography

Dr. Rathore is an associate professor of Pediatrics and Neurosciences at University of Nebraska Medical Center, Omaha, Nebraska, USA. She completed medical school from Sampurnanad Medical School in India and completed her Pediatric Neurology Training in Child Neurology from Baylor college of Medicine and Texas Children's Hospital, Houston, USA in 2015. Dr. Rathore has an experience of treating patients with Pediatric Autoimmune encephalitis for over 10 years and has published many abstracts, articles and book chapters in the field of Neuroimmunology. She has presented her work at many national and international meetings and is a considered an international expert in the condition.

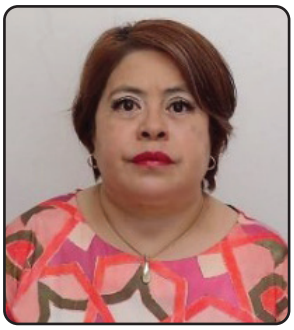
Benjamin Pinker*, Anna-Maria Barciszewska
Karol Marcinkowski University of Medical Science, Poland

mTOR signaling and potential therapeutic targeting in meningioma

Meningiomas are the most frequent primary tumors arising in the central nervous system. They typically follow a benign course, with an excellent prognosis for grade I lesions through surgical intervention. Although radiotherapy is a good option for recurrent, progressive, or inoperable tumors, alternative treatments are very limited. mTOR is a protein complex with increasing therapeutical potential as a target in cancer. The current understanding of the mTOR pathway heavily involves it in the development of meningioma. Its activation is strongly dependent on PI3K/Akt signaling and the merlin protein. Both factors are commonly defective in meningioma cells, which indicates their likely function in tumor growth. Furthermore, regarding molecular tumorigenesis, the kinase activity of the mTORC1 complex inhibits many components of the autophagosome, such as the ULK1 or beclin complexes. mTOR contributes to redox homeostasis, a vital component of neoplasia. Recent clinical trials have investigated novel chemotherapeutic agents for mTOR inhibition, showing promising results in resistant or recurrent meningiomas.

Keywords: meningioma; mTOR; redox homeostasis; macroautophagy; everolimus; vistusertib; lycopene

VIRTUAL



Ana Lilia Rodriguez Villegas
Programa de Doctorado en Psicologia, Universidad Nacional Autónoma de México, Ciudad de Mexico, State, México

Executive function and adaptive coping as frontal lobe functions in healthy adults

This study evaluated the relationship between executive function and Adaptive Coping Strategies (ACS) in healthy adults between the ages of 43 and 52. The participants (n=104) 52 males and 52 females had no history of neurological or psychiatric illnesses or chronic conditions. Neuropsychological assessment was performed with Tower of London DX (TOLDX) test, the Wisconsin Card Sorting Test (WCST) and the Coping Strategies Inventory. A relationship was found between the percentage of error and conceptual-level responses (both WCST) and the Problem Solving (ACS). In a separate analysis performed on the males, a negative relationship was identified between the WCST responses and the Emotional Expression (ACS). In the female group the dimensions of the WCST and the TOLDX were associated with the emotional Expression and Problem Solving ACS subscales and the maladaptive coping strategy Social Withdrawal subscale. The relationship between executive function and ACS is multidimensional and complex.

Among the most important influences in this relationship, it was found the gender, probably as a consequence of cultural learning. Importantly, the critical anatomical area it seems to be the Dorsolateral Prefrontal Cortex (DLPFC) that is related with specific aspects of executive functions. Finally, If the stressing situation was related to changes in the diary routine, better was the executive function performance, possibly this can be explained by both emotional and cognitive aspects, specifically self-confidence and flexibility cognitive.

Using a neuropsychological analysis with ecological validity, this study adds the view of frontal lobe socio-cultural plasticity. Future research should consider designer studies with focal neurological disorders.

Biography

Dr. Ana Lilia Rodríguez Villegas studied a Psychology Bachelor (1992-1996) a Mater Degree in Neuropsychology (1998-2000) and a Psychology PhD (2011-2015) at National Autonomous University of México. She received her PhD degree, in 2014 in the same institution, with an acknowledge for early graduation. She was working in public health system particularly with neurosurgical patients and lysosomal storage disorders with neuropsychological assessments. She has an international article in Applied Neuropsychology Adult. She is member of International Neuropsychological Society and Canadian Psychology Association.

VIRTUAL

Tongtong Li*[1], Yu Zheng[1], David Zhu[2], Jian Ren[1], Taosheng Liu[3], Karl Firston[4]

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[4]The Wellcome Centre for Human Neuroimaging, University College London, London, UK

Brain information processing capacity modeling

Characterizing the information processing capacity of the human brain is a key challenge in cognitive psychology and neuroscience. Most of the existing research in this area has focused on the capacity limit of short-term working memory, or how well an individual handles information processing demands when several tasks have to be executed simultaneously. It is believed that our visual short-term memory can maintain representations of three to four objects at any given moment. Along this line, information processing capacity was mapped to the computational capacity of a dynamic system and characterized as the total number of linearly independent functions of its stimuli the system can compute.

Previous research in neurophysiology suggests that human information processing is reflected in neuronal activity. Existing models of neuronal activity offer a panoramic coverage of brain dynamics, from the single neuron, through neural populations, to brain networks. However, under all these models, the quantitative relationship between the activity of a brain region and its information processing capacity remains unclear.

In this paper, we considered neuronal activity and information processing capacity from an information-theoretic perspective. Starting from an information conservation law, we showed that for an individual brain region, the neuronal activity, the information processing capacity, the input storage capacity, and the arrival rate of exogenous information can all be related through a first-order differential equation. Theoretically, our model indicates that the difference between the information arrival rate and the information processing rate directly influences neural activity changes. Higher information arrival rate enhances the neuronal activity, while larger processing capacity decreases neuronal activity; on the other hand, larger input information storage capacity can alleviate the demand on neuronal activity, when the arrival rate increases.

We applied this model to an empirical fMRI dataset, which was acquired under a rapid event-related arrow flanker task—used to study aging-associated decline in selective attention and executive functions. Both young and old adult groups participated in the experiment. We analyzed individual brain regions that were activated in both the young and old groups. We also considered overall information processing by averaging the data from each region. Our numerical analysis demonstrated the accuracy of the model in explaining fMRI measurements and showed that—for a given cognitive task—higher information processing capacity engenders a lower neuronal activity and faster response in younger subjects. That is, younger adults have faster responses and better performance in the flanker test than the seniors because they have higher information processing capacity. This result is consistent with the findings in literature suggesting that high-capacity individuals tend to have lower neuronal activity, and that—compared with young adults—more brain activation was required for older adults to accomplish the same cognitive task. Crucially, these findings speak to the predictive and construct validity of the model, in the sense that we were able to predict the behavioral responses more accurately from (independent) fMRI responses.

While the processing capacity model is a new finding, it is reassuring that—although originating from information theory—our model has a similar functional form to the conductance-based neural mass models

in DCM, as well as the IF model of individual neurons. The implication here is that—with an information conservation law as the cornerstone—our model is not limited to brain regions but can be applied to any neuronal system that has the attributes of information processing and storage capability. In sum, the model offers a framework for multiscale modelling of brain dynamics in terms of information processing and provides a new perspective on computational architectures in the brain; and it can be applied to any data from which neuronal activity can be estimated.

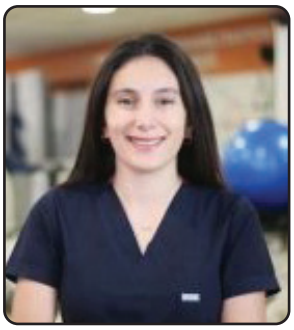
Audience Take Away:

- The audience will learn an innovative method on how to estimate localized information processing and storage capacity from neuronal activity in individual brain regions or brain networks.
- This paper is an initial step towards the quantitative characterization of the information processing capacity of individual brain regions. The IPC model offers a framework for modelling of brain dynamics in terms of information processing capacity, and may be exploited for studies of predictive coding and Bayes-optimal decision-making. The model can be used to explore the capacity limit of human brain, and can also be used to evaluate the information loss in different neural systems or brain regions, especially those involved in overflow-driven faulty decision making or abnormal conditions such as Alzheimer's disease or seizures.

Biography

Tongtong Li is a Professor at Department of Electrical and Computer Engineering, Michigan State University. Prof. Li's research interests fall into the areas of communications, information theory and statistical signal processing, and brain network analysis using communication theory, with applications to Alzheimer's disease and related research. Taking the brain as a communication network, she has been working on the modeling and analysis of brain information processing capacity, input storage capacity, neuronal activity, functional connectivity, causality, stability, and the impact of age and cognitive impairment on brain network functions and performances.

VIRTUAL



Saral İlknur[1,2], Yayla Yasemin Tugce*[2], Çakar Engin[2,3]

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[2]Department of Physical Medicine and Rehabilitation, Memorial Hospital, Turkey
[3]Department of Physiotherapy and Rehabilitation, Faculty of Health Sciences, Üsküdar University, Turkey

A retrospective exploration of in-hospital rehabilitation findings in post-stroke patients: Functional measures and complications

Background: Stroke is a disabling disease due to its functional impairments and complications. In this study, we aimed to examine the clinical profiles, practical outcomes, and complications during rehabilitation of patients hospitalized with stroke.

Methods: In this single center, retrospective study, 49 patients who experienced stroke (post-stroke time range, 1–72 months; mean age 56 ± 13.17 years; sex, 40 men and 9 women; stroke etiology, 61.2% ischemic; hemiplegic side, 57.1% left; median length of hospital stay, 102.69 ± 86.17 days) were assessed between January 03, 2020 and January 03, 2022. Comparisons and correlations between Brunnstrom motor staging, Functional Ambulation Scale, and Barthel Index of activities of daily living scores as well as complications recorded at hospital admission, stay and discharge were statistically analyzed.

Results: A statistically significant improvement in Brunnstrom motor staging, Functional Ambulation Scale, and Barthel Index scores was observed at discharge compared to those at hospital admission ($p<0.001$). The most common complications during inpatient rehabilitation were urinary tract infections, pneumonia, decubitus, and conjunctivitis (75.51%, 38.78%, 18.37%, and 10.2%, respectively). Correlation analysis showed a significant negative correlation between the improved Functional Ambulation Scale and Barthel Index scores and decreased risk of complications (Spearman's rho $r = -0.419$ and $r = -0.565$ for pneumonia; $r = -0.362$ and $r = -0.305$ for decubitus; $r = -0.299$ and $r = -0.380$ for conjunctivitis, respectively).

Conclusion: The results of this study suggest that improvement in functional measures is essential during rehabilitation to decrease the risk of complications in patients post stroke. We believe that management, monitoring, and follow-up of post-stroke patients are high-priority issues to consider.

Biography

Yasemin Tugce Yayla. studied at East Mediterranean University. He have experience in Yağmur Ozer Education Center in Bursa. He have been working at Memorial Şişli Hospital since 2017. He have an internship in Ordu Medical Park Hospital between 2012-2016. He participated in proprioception and education course 5-9 November 2014. He have a diabetic foot assessment and cad-cam underlay application certificate between 5- 9 November 2014. He have an internship at East Meditterrian University between 2015-2016. I have an internship in Gazimagusa Government Hospital between 2015- 2016. He have an internship at Hacettepe University Hospital between 1 July- 22 August 2016. He have attended the first global Turkish physical therapy and rehabilitation congress from 8-9 April 2016.

VIRTUAL

Michelle Herman
Inspirational Speaker, Michelle Herman Speaks, Sacramento, California, United States

Inside the mind of a 32 year old stroke survivor living with moya moya disease

At the age of 32, and 6 months postpartum from having her first child, Michelle walked into the ER with impaired motor skills on the left side of her body, a horrible headache and slurred speech. She was diagnosed with a migraine and discharged. Two days later she had a massive Ischemic Stroke. The cause of her stroke went undetected for more than 6 months, until she was finally diagnosed with moya moya disease and underwent brain surgery to restore blood and oxygen to the right side of her body. Michelle now shares her story with medical professionals hoping she can help to save another person from the suffering she went through.

Audience Take Away:

- They will learn the major symptoms of a TIA and how it differs from a migraine, the next time someone presents with a TIA, they will know what to do and how to help before the patient has a full stroke. They can save a life with this information
- They will hear first-hand, what it is like to be the patient having a stroke and what it is like to have a brain injury so they can provide better care for their patients moving forward

Biography

Michelle Herman is a charismatic and engaging award-winning Public Speaker and Communications professional with an undeniable passion for showing compassion and empathy to others. Michelle's medical journey in her early 30's consisted of a TIA being misdiagnosed, leading to a major stroke and eventually a moya moya disease diagnoses, a lifesaving brain surgery and chronic depression. Michelle openly shares her story and struggles with the medical community, to give insight to the patient experience.

VIRTUAL

Annapurna Ahuja
Department of Periodontics and Oral Implantology, Hazaribag college of dental sciences and Hospital/ Affiliated to Dental council of India, Hazaribag, Jharkhand, India

Periodontitis and neurodegenerative disorders. A possible Link

Neurodegenerative diseases are a group of progressive disorders that affect the Central Nervous System (CNS) such as Alzheimer, Parkinson, and multiple sclerosis. Inflammation plays a critical role in the onset and progression of these injuries. Periodontitis is considered an inflammatory disease caused by oral biofilms around the tooth-supporting tissues, leading to a systemic and chronic inflammatory condition. Among the different types of neurodegenerative diseases, Alzheimer's Disease (AD), Huntington's disease, Parkinson's Disease (PD), and multiple sclerosis are the most frequently occurring (Hussain et al., 2018). These disorders damage the CNS and trigger rapid microglial activation, the main component of neuroinflammation. Activated microglia produce and secrete inflammatory mediators, such as eicosanoids, cytokines, chemokines, reactive free radicals, and proteases. Although a well-regulated inflammatory process is beneficial for injured CNS tissue, an excessive inflammatory response can be a source of additional injury and may affect the chronic progression of these diseases (Gao and Hong, 2008).

Robust evidences has indicated a correlation between PD and systemic diseases such as cardiovascular disease, adverse pregnancy outcome, chronic kidney disease and diabetes mellitus. However, the correlation between teeth loss and the risk of neurodegenerative disorders are still under investigation. The conditions of periodontal disease (bacterial infection and chronic inflammation) weaken the blood-brain barrier and pose a risk for cerebrovascular disease. The conditions also cause inflammation in the brain. Periodontal disease may indirectly make neurodegenerative disorders worse through exacerbation of diabetes. Furthermore, tooth loss leads to deterioration of cognitive function. Therefore, it is possible that periodontal disease directly and indirectly exacerbates the condition of these disorders.

Audience Take Away:

- Learning the importance of oral hygiene and effects of periodontitis on systemic health
- Audience shall learn about the effects of oral microorganisms on nervous system
- Along with seeking treatment for neurodegenerative diseases one should consider visiting oral health specialist

Biography

Professor Dr. Annapurna Ahuja, studies her Masters of dental surgery from the prestigious college of dental science and hospital, affiliated to Rajiv Gandhi University of health sciences, Bangalore in 2009. She then joined Dental College as a Teaching Faculty. She has published more than 40 research articles, 8 text books of her specialty, one chapter contribution. She is a very active part of the research and has 4 copyrights of her work in the field of Periodontics. She is also a renowned poet, has written more than 200 poems. Has a recipient of Best book award for her debut book, BAPUJI written in Indian Language. Presently she is associated with Hazaribag College of Dental Sciences & Hospital, as Head of the Department, Dept. of Periodontics & Oral Implants.

Almutazballlah Bassam Qablan*[2], Yasmeen Jamal Alabdallat[1], Hamza Al-Salhi[3], Salameh Alarood[4], Ibraheem Alkhawaldeh[5], Obada Abunar[6], Adam Abdallah[7]

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[3]Faculty of Medicine, Hashemite University, Jordan Amman
[4]Medical doctor, Jordan Alkarak
[5]Faculty of Medicine, Hashemite University mutah university, Jordan Alkarak
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Hypoglossal nerve stimulation (baseline vs. 12 months) for obstructive sleep apnea: A meta-analysis

Background: Obstructive Sleep Apnea (OSA) is a disorder caused by the repeated collapse of the upper airway during sleep. It is the most common cause of sleep-related breathing disorder, as OSA can cause loud snoring, daytime fatigue, or more severe problems such as high blood pressure, cardiovascular disease, coronary artery disease, insulin- resistant diabetes, and depression. The Hypoglossal Nerve Stimulator (HNS) is an implantable medical device that reduces the occurrence of obstructive sleep apnea by electrically stimulating the hypoglossal nerve in rhythm with the patient's breathing, causing the tongue to move. This stimulation helps keep the patient's airways clear while they sleep. This systematic review and meta-analysis aim to assess the clinical outcome of hypoglossal nerve stimulation as a treatment of obstructive sleep apnea.

Methods: A computer literature search of PubMed, Scopus, Web of Science, and Cochrane Central Register of Controlled Trials was conducted from inception until August 2022. Studies assessing the following clinical outcomes (Apnea-Hypopnea Index (AHI), Epworth Sleepiness Scale (ESS), Functional Outcomes of Sleep Questionnaire (FOSQ), Oxygen Desaturation Indices (ODI), (Oxygen Saturation (SaO2)) were pooled in the meta-analysis using Review Manager Software. We assessed the quality of studies according to the Cochrane risk-of-bias tool for randomized trials (RoB2), Risk of Bias In Non-randomized Studies - of Interventions (ROBINS-I), and a modified version of NOS for the non-comparative cohort studies.

Results: 13 Studies (Six Clinical Trials and Seven prospective cohort studies) with a total of 817 patients were included in the meta-analysis. The results of AHI were reported in 11 studies examining OSA 696 patients. We found that there was a significant improvement in the AHI after 12 months of HNS (MD = 18.2 with 95% CI, (16.7 to 19.7; I2 = 0%); P < 0.00001). Further, 12 studies reported the results of ESS after 12 months of intervention with a significant improvement in the range of sleepiness among the examined 757 OSA patients (MD = 5.3 with 95% CI, (4.75 to 5.86; I2 = 65%); P < 0.0001). Moreover, nine studies involving 699 participants reported the results of FOSQ after 12 months of HNS with a significant reported improvement (MD = -3.09 with 95% CI, (-3.41 to 2.77; I2 = 0%); P < 0.00001). In addition, ten studies reported the results of ODI with a significant improvement after 12 months of HNS among the 817 examined patients (MD = 14.8 with 95% CI, (13.25 to 16.32; I2 = 0%); P < 000001).

Conclusion: The Hypoglossal Nerve Stimulation showed a significant positive impact on the obstructive sleep apnea patients after 12 months of therapy in terms of apnea-hypopnea index, oxygen desaturation indices, manifestations of the behavioral morbidity associated to obstructive sleep apnea and functional status resulting from sleepiness.

Keywords: Apnea, Hypoglossal, Meta-Analysis, Stimulation.

Audience Take Away:

- We will talk about the pioneering method of hypoglossus nerve stimulation and its effectiveness, as studies have shown its efficiency and effectiveness as a treatment for OSA patients in tolerated to CPAP
- Studies also report subjective improvement in measures of quality of life and continued efficacy of HGNS in the same patients after four years
- HGNS status is indicated for some patients with moderate to severe obstructive sleep apnea, as determined by an AHI score of 15 to 65. HGNS is currently being studied as an alternative to CPAP for children and Down syndrome adolescents. According to the Food and Drug Administration, people who have previously failed or could not tolerate treatment with positive airway pressure therapies are good candidates for HGNS. In addition, not everyone with obstructive sleep apnea shows the same airway anatomy and tongue collapsibility pattern during sleep. People who sleep with some anatomical patterns are better suited to HGNS than others
- While CPAP is often recommended as a first-line treatment due to its success, inconsistent use may be caused by the intrusive feel of the mask, set-up time, and cost of the device and parts. Some surgical techniques have been used in OSA patients. Unfortunately, conventional methods of surgery did not improve obstructive sleep apnea in 59% of patients. Unlike other surgeries, HGNS does not require permanent removal or alteration of airway tissue. The hypoglossal nerve is also located anatomically for convenient surgical access and is responsive to stimulation

Biography

Dr. Almutazballlah Qablan, 6th year medical student at Jordan University of Science and Technology, Class representative at Jordan University of Science and Technology, Medical script writer at Prints and More medical Team, Medical script writer at Neuropedia Team, Aspiring Student in Neurology.

VIRTUAL

Paulo Henrique Leite Souza*[1], Joao Carlos Ferrari Correa[1], Laura Uehara[1], Raphael Mendes Ritti Dias[1], Gustavo Oliveira da Silva[1], Kevin Pacheco-Barrios[2], Felipe Fregni[3], Fernanda Ishida Correa[1]

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[2]Neuromodulation Center and Center for Clinical Research Learning, Spaulding Rehabilitation Hospital and Massachusetts General Hospital, Harvard Medical School, Boston, USA. Universidad San Ignacio de Loyola, Vicerrectorado de Investigación, Unidad de Investigación para la Generación y Síntesis de Evidencias en Salud. Lima, Peru
[3]Neuromodulation Center and Center for Clinical Research Learning, Spaulding Rehabilitation Hospital and Massachusetts General Hospital, Harvard Medical School, Boston, USA

Effect of transcutaneous auricular vagus nerve stimulation on inflammation, cardiac autonomic modulation and health status of individuals with COVID-19: A randomized, blind, and controlled trial

Introduction: COVID-19 has different clinical symptoms, with excessive inflammation and cardiovascular changes being common in severe cases. The use of transcutaneous atrial Vagus Nerve Stimulation (taVNS) seems to be a viable treatment for these alterations.

Objective: To evaluate the effects of transcranial auricular Vagus Nerve Stimulation (taVNS) on inflammation, cardiac autonomic modulation, and clinical evolution of individuals affected with COVID-19.

Method: Clinical, controlled, blinded, randomized trial, in which 52 adult subjects, hospitalized with COVID-19, were randomized to receive active taVNS (a-taVNS) or sham taVNS (s-taVNS). The a-taVNS was applied for 90 minutes, twice a day, for 7 days, totaling 14 sessions, for the s-taVNS, the device remained off. Pre- and post-intervention inflammatory mediators were evaluated: interleukin-6 (IL-6), interleukin-10 (IL-10), C-reactive protein (CRP), and cortisol and Heart Rate Variability (HRV). Clinical evolution, which included clinical signs of the disease and levels of anxiety and depression, was evaluated pre-and post-intervention and at 7 and 14-day follow-up. Attention and memory levels were also monitored after 7 and 14 days and monthly for 6 months, after the end of the interventions.

Results: There was a significant reduction in CRP ($p=0.038$) and IL-6 ($p<0.001$) for the experimental group. There were no changes in IL-10 and cortisol levels and in HRV results ($p>0.05$) in both groups. In the clinical evolution, there were no changes in the variables in the evaluated periods, except for a significant decrease in the level of depression ($p=0.031$) in the a-taVNS group.

Conclusion: taVNS showed effects on inflammatory mediators CRP and IL-6 and on depression levels, however, it had no effects on cardiac autonomic modulation and other clinical symptoms.

Keywords: COVID-19, Vagus Nerve Stimulation, inflammation, autonomic modulation, clinical course, heart rate variability.

Audience Take Away:

- Symptoms of COVID-19 are highly variable and can affect the respiratory, cardiac, neurological and other systems, resulting from a hyperinflammatory state known as a cytokine storm
- The vagus nerve participates in the regulation of important homeostatic functions and studies have confirmed its participation in the modulation of inflammation; the understanding of this pathway reveals an important therapeutic role of vagus nerve manipulations
- Vagus nerve stimulation has been investigated as a promising therapy for inflammatory processes, including COVID-19 and cardiovascular disorders

- There is evidence that vagus nerve stimulation can decrease sympathetic activity and is associated with the release of norepinephrine, improving autonomic control

Biography

Physiotherapist at Nove de Julho University (UNINOVE, Brazil). I was a Scientific Initiation student with a PIBIC grant under the guidance of Professor Fernanda Ishida Corrêa in the area of Neuromodulation. I am currently studying for a Masters in Rehabilitation Sciences at the same institution with a CAPES grant under the guidance of Professor Fernanda Ishida Corrêa and Professor João Carlos Ferrari Corrêa. I am member of the Scientific Discussion Group from the Laboratory of Neuromodulation, Functionality and Movement Analysis (LANFAM).

VIRTUAL

Priyanka Sethi
GD Goenka University, India

Prevalence and diagnosis of fibromyalgia: A narrative review

Background: Fibromyalgia (FM) is a chronic disorder with persistent manifestation of pain, mood swings, fatigue, depression and attention deficits that affects one's activity of daily living.

Objective: The present study aimed to find prevalence and diagnosis of Fibromyalgia.

Methodology: A systematic review was conducted to check the prevalence of fibromyalgia from 2011 to 2020, where records were taken from MEDLINE, PUBMED and SCOPUS, in total of 3274 records were identified. Studies were selected based on the selection criteria to check the prevalence of fibromyalgia worldwide. The Studies associated with any other disease were excluded. The obtained studies were further divided into five groups based on the key words. "Prevalence of Fibromyalgia", "Fibromyalgia and Dysmenorrhea", "Fibromyalgia and Anxiety, Depression, Demography", "Chronic pain and fibromyalgia", "Fibromyalgia and Gender" categories. Five studies have revealed the highest incidence of fibromyalgia in women with age group 29-50 years of any demography along with the associated symptoms of anxiety, depression, and behavioural changes. This narrative review has shown a substantial growth in the cases of fibromyalgia in an expanded population.

Conclusion: This study concluded that there is a significant prevalence of symptoms of fibromyalgia among the population of any age and gender but the criteria used by various researchers need further need more extensive review to find out the actual evidence of fibromyalgia in the community worldwide. Further the literature review yet need to set and include some other associated symptoms along with fibromyalgia so that the prevalence of this condition can further be evaluated extensively.

Key Words: Fibromyalgia, Anxiety, Depression, Demography, Chronic pain

Biography

Dr. Priyanka Sethi is working as Assistant Professor, Department of Physiotherapy, Faculty of Allied Health Sciences, MRIIRS. She has a rich experience of 13 years in the field of Physiotherapy practice majorly in Academics, Clinical and Healthcare IT (Health fore Technologies) - the first Healthcare domain in North India with PAN India. Beside this, she has worked in various health related projects like Ergonomics, and Diet counselling for diabetic patients for the Fortis Hospital. She has done her Graduation in 2007 from Guru Nanak Dev University (Punjab) and Masters in Physiotherapy IN 2009 from Lovely Professional University Phagwara (Punjab) with Specialization in Advanced Neurological Conditions. She is pursing PhD in Physiotherapy from GD GOENKA University, Gurugram. She has published various national and international papers in journals of repute and attended various national and international conferences. She has filed many copyrights and patent publications in Intellectual property rights. She has been awarded with Best Professor award in 2021. She has a key interest areas like Lifestyle management for Elderly, Stroke Prevention and management and Women Health

VIRTUAL

Bruany Antoniolli Bianchi*, Mario Vicente Campos Guimaraes, Ana Beatriz Lima Pedroza, Natália Moreno Coelho, Josue Andrade Martins, Julia Souza e Costa

Anhembi Morumbi University, Sao Paulo - SP, Brazil

Hypoxic-ischemic encephalopathy secondary to fat emboli responding to modafinil treatment: A case report and brief literature review

Background: Fat Embolism Syndrome (FES) is associated with long bone fractures, occurring with a variable frequency of 0.5 to 3.5% after surgical treatment. It affects 90% of open fractures of long bones, being 75% femoral and 19% tibial fractures. Hypoxic - ischemic encephalopathy is a serious condition resulting from low brain perfusion, FES is a possible cause that leads to intraparenchymal damage.

Objectives: This paper aims to report a successful hypoxic-ischemic encephalopathy treatment using modafinila.

Methods: The case reported in this paper was gathered by appreciation of the patient's records and interviews and was discussed in the light of the most recent literature.

Clinical Presentation: A 26-years-old man comes to the Guarulhos Municipal Urgency Hospital after a femoral and tibial injury caused by a motorcycle accident. At admission, he was conscious and oriented. The next day, he evolved into tachypnea, sweating, low conscious level, answering only to painful stimuli, presenting fat emboli syndrome. Succeeding the inferior member's external fixation surgery, in the Intensive Care Unit (ICU), the patient went through septic shock with pulmonary foci, pulmonary thromboembolism and three cardiopulmonary arrest. Following the hemodynamic and infectious improvement, the patient progressed to hospital discharge after 40 days. At that time, he presented discrete conscious level and motor improvement. Afterwards, the patient manifested seizures and hydrocephalus was diagnosed. He underwent ventriculoperitoneal shunt surgery, later replaced by external ventricular drain, evolving to post-surgery infectious meningitis. In the permanence of seizure, it was prescribed sodium valproate. Then, modafinil, 100 mg/day, was introduced in the treatment. The patient progressed with improvement of scapular waist, head sustentation, cognitive performance, memory, verbal communication and also was capable of deambulation. Sodium valproate was suspended, as the patient presented progressed.

Discussion: In fat embolism patients, neurological injury is a possible outcome, occasionating hypoxic-ischemic encephalopathy, a condition caused by brain low perfusion. Our patient presented motor and conscious impairment after a femoral and tibial injury that showed improvement by the modafinil administration. This case is in consonance with the literature reports which demonstrates cognition, attention and motor response to the treatment. However, it is still not fully clear which action mechanisms modafinil uses for this matter. Modafinil is an alpha1B noradrenergic receptor stimulant, increasing glutamate and histamine release and decreasing Gamma-AminoButyric Acid (GABA), reducing apoptosis and gliosis, comprising possible mechanisms for neuronal protection.

Conclusion: Modadinil is a promising treatment for cerebral injuries, promoting cognitive improvement. However, further studies are required to clarify its action mechanism. The use of modadinil for hypoxic - ischemic encephalopathy

Biography

Bruany Antoniolli Bianchi, journalist and medical student. Worked in Journal Globo in 2018. Today, she is a fourth-year medical student. Founder and Researcher in Duchenne Dystrophy Institute and Memory Institute. Chapter publisher about transcranial stimulation in neuropsychiatry diseases. Anhembi Morumbi University Neurology and Neurosurgery group studies director. Clerkship at Neurosurgery Department of Guaxupe's Santa Casa.

VIRTUAL

Lama Saad El Din Mahmoud
Depatment of Neurology and Neurosurgery, October 6 University, Faculty of Physical Therapy, Lecturer of Neurology and Neurosurgery, Giza, Egypt

Effect of motor learning augmented cues on cognitive functions in parkinson

Background/Aims: Parkinsonism characterized by deficits in cognitive functions that affect the motor control. The main aim was to assess the effect of motor learning feedback with augmented cues of motor learning on cognitive functions and motor performance in patients with parkinsonism.

Methods: A total of 30 patients from both sexes participated in this study. The patients were randomly divided equally into two equal groups: the study group received motor learning feedback with augmented cues of motor learning and the conventional treatment. The control group received conventional cognitive training. The patients assessed by the computer-based cognitive assessment device (RehaCom).

Findings: The result of this study showed that there was a significant difference between the study and control groups (p=0.0001), which indicated that the study group showed a greater improvement than the control group.

Conclusions: The motor learning feedback with augmented cues of lead to significant improvement effect on both cognitive and motor functions in patients with parkinsonism.

Key words: Cognition, Motor learning, Parkinsonism.

Audience Take Away:

- The audience will be able to practice the motor learning in treatment of neurological disorders
- The audience will get new advanced knowledge on how to apply the motor learning feedback in rehabilitation program in Parkinson patients
- Motor learning therapy have a significant effect in improving cognitive and functional outcomes in neurological disorders

Biography

Lama Saad El-Din Mahmoud has been currently working as lecturer of physical Therapy for Neuromuscular disorders & its surgery, faculty of physical therapy, October 6 university, Egypt and working as Neurology and Neurosurgery Consultant for Neuro-Rehabilitation. Ph.D. degree, Department of Physical Therapy for Neuromuscular Disorders and its Surgery, faculty of physical therapy, Cairo university

VIRTUAL

Esraa Askar*, Harsimran Gill, Neeraj Singh
Department of Internal Medicine, Northwell/Hofstra Zucker School of Medicine, Long Island Jewish Forest Hills, NY, USA

A case of transient global amnesia triggered by sexual intercourse

Introduction: Transient Global Amnesia (TGA) involves a sudden onset of anterograde and retrograde amnesia, often accompanied by executive dysfunction, typically lasting up to 24 hours, and not linked to another neurological impairment.

Case Presentation: A 52-year-old right-handed man presented with confusion and retrograde amnesia starting during the morning of presentation. He had been driving on a long road trip the previous day without alteration of consciousness, but subsequently forgot a period of a few hours during the trip. On initial evaluation in the Emergency Department, he had poor short-term memory and could not state the current year or president, nor his home address. He had no signs of stroke on exam. He denied any chronic medical conditions or medications. He notably experienced headache during sexual intercourse after the trip and before he first complained of amnesia. A CT brain scan four hours after onset of amnesia revealed no acute intracranial abnormalities. A MRI brain scan 13 hours after onset of amnesia demonstrated only a right hippocampal punctate focus of diffusion restriction. On exam 24 hours after presentation, the patient had full short-term recall, and was oriented to person and place, but not to date.

Discussion: The cause of TGA is uncertain despise its widespread recognition as a clinical entity. It has been linked to vascular risk factors, migraine, and epilepsy. Bilateral hippocampal ischemia has been hypothesized as a probable cause of transient loss of declarative memory, while migraines and epilepsy have been suggested as possible TGA correlates.

Conclusion: While TGA is uncommon and has a favorable prognosis, it can be seen in typically healthy individuals with no medical comorbidities. This case reveals the presence of sexual intercourse as a probable trigger for TGA, or alternatively a trigger for new-onset migraine which itself is a known TGA correlate. This unique presentation of TGA can guide clinicians in the future on identifying relevant risk factors for an accurate diagnosis of TGA.

Audience Take Away:

- Describe the typical clinical features of transient global amnesia
- Identify typical neuroimaging findings associated with transient global amnesia
- Outline the differential diagnoses and clinical correlates for transient global amnesia

Biography

Dr. Askar graduated medical school at Faculty of Medicine, Alexandria University, Egypt in 2016. She then worked as a teaching assistant at Microbiology and Immunology Department Faculty of Medicine, Alexandria University for 2 years, during which she finished her USMLE exams and then joined the Internal Medicine Residency training program at Department of Internal Medicine, Northwell/Hofstra Zucker School of Medicine, Long Island Jewish Forest Hills, NY, USA. She was honored to be the intern of the year during her first year of residency 2020-2021. She then was chosen by her program to be the chief resident during her third year of residency 2022-2023.Throughout her medical school, she was a member of “Future Doctor Society” which is a non-governmental organization in Egypt aiming to improve the health care system. She participated in many fundraising projects, campaigns, and social events.

VIRTUAL

Esraa Askar*, Ramy Abukwaik, Regine Cherazard
Department of Internal Medicine, Northwell/Hofstra Zucker School of Medicine, Long Island Jewish Forest Hills, NY, USA

Aortic graft infection secondary to campylobacter fetus

Introduction: Early aortic graft infections, which can arise within 4 months of endovascular graft repair, frequently present as high-grade infections. However, late infections can be challenging to detect due to nonspecific symptoms. This case report discusses a late aortic graft infection developing 3 years after an endovascular abdominal aortic aneurysm repair (EVAR).

Case Description: A 75-year-old man who underwent EVAR 3 years prior to presentation reported generalized weakness, cough, and rhinorrhea lasting 4 days. On admission, the patient was febrile (39.3 °C), with a pulse rate of 95 beats/min, respiratory rate of 18 breaths/min, and 93% oxygen saturation on room air. His blood pressure was within reference range, and his physical examination and chest radiography findings were unremarkable. His viral assays and urinalysis were negative. His laboratory evaluation revealed a white blood cell count (WBC) of 12.63 × 109/L (reference range, 3.8–10.5 × 109/L). His abdominal Computed Tomography (CT) scan revealed perianeurysmal stranding. Blood cultures collected on admission grew gram-negative rods within 2 days, and the patient was treated with ceftriaxone.

Campylobacter fetus as the causative agent increased suspicion of aortic graft infection. His antimicrobial management escalated to ertapenem, and the care team consulted the vascular surgery department. A CT angiogram of the abdominal aorta using an intravenous contrast agent revealed a type II endoleak, periaortic stranding, and lymph nodes. A whole-body indium scan showed increased leukocytic accumulation in the periaortic region, indicating infection. The patient was transferred to his primary surgeon for Positron Emission Tomography (PET)-CT examination and further disease management.

Discussion: Aortic endograft infection is a rare complication following EVAR, appearing in <1% of cases but more frequently after emergency or repeat surgical operations due to intraoperative bacterial contamination. Remarkably, this patient experienced an aortic graft infection 3 years after EVAR in Campylobacter fetus bacteremia. The high affinity of a surface receptor for vascular tissue- especially damaged endothelium and the production of a local procoagulant promoting thrombus formation have been associated with the vascular tropism of Campylobacter fetus. Vascular graft infections require a timely diagnosis for appropriate surgical and/or antibiotic treatment to reduce mortality. Unnecessary surgical intervention on non infected grafts is associated with high mortality risk, making the accurate diagnosis of vascular graft infections imperative.

The challenge with a clinically suspected vascular graft infection is obtaining conclusive evidence. While difficult to obtain in clinical practice, positive cultures from percutaneously aspirated perigraft fluid or surgically retrieved material are the gold standard for determining the diagnosis. Furthermore, most clinical signs and symptoms are nonspecific. WBC scintigraphy with single-photon emission computed tomography/CT has high diagnostic accuracy but is time-consuming. Fluorodeoxyglucose- PET/CT examination is preferred for an expeditious diagnosis.

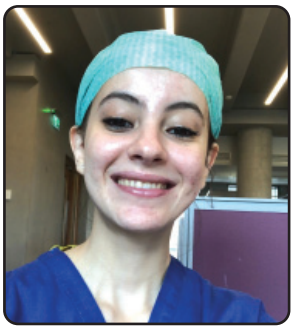
Audience Take Away:

- Describe the non-specific clinical features of aortic graft infection
- Introducing Campylobacter fetus as a causative agent of aortic graft infection
- Outline different modalities for diagnosis of aortic graft infection

Biography

Dr. Askar graduated medical school at Faculty of Medicine, Alexandria University, Egypt in 2016. She then worked as a teaching assistant at Microbiology and Immunology Department Faculty of Medicine, Alexandria University for 2 years, during which she finished her USMLE exams and then joined the Internal Medicine Residency training program at Department of Internal Medicine, Northwell/Hofstra Zucker School of Medicine, Long Island Jewish Forest Hills, NY, USA. She was honored to be the intern of the year during her first year of residency 2020-2021. She then was chosen by her program to be the chief resident during her third year of residency 2022-2023. Throughout her medical school, she was a member of “Future Doctor Society” which is a non-governmental organization in Egypt aiming to improve the health care system. She participated in many fundraising projects, campaigns, and social events.

VIRTUAL



Fareha Khalil*[1], Suzanne Murphy[2]
[1]University of Limerick, Limerick, Ireland
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Developmental outcome and peri-natal diagnosis of agenesis of the corpus callosum

Background and aim: Agenesis of the corpus callosum can lead to varied neurodevelopmental outcomes. The prevalence of isolated Agenesis of the Corpus Callosum (iACC) is not known; selection bias in reported series as well as changes in how ACC was diagnosed prenatally, and lack of consensus on the best threshold to diagnose iACC means it is possible a significant number of individuals with iACC have escaped detection.

The study aim was to systematically review the evidence published on the developmental outcomes of a pre-natal diagnosis of isolated ACC and review the peri-natal diagnostic methods.

Method: A systematic search using the electronic databases PubMed, Cochrane, and Medline was carried out. The search was restricted to English language publications in peer-reviewed journals. PRISMA guidelines were followed. Seventeen studies met the inclusion criteria.

Results: Pooled rates of neurodevelopmental delay were 29.6% (95% CI, 24.8-34.3) and normal outcome was 70.3% (95% CI, 66.2-74.3) in iACC (figure 1). Post-natal MRI detected additional abnormalities in 67/207 (31.4%; 95% CI, 24.7-38) apparently isolated cases.

Conclusions: A prenatal diagnosis of isolated ACC may in fact carry a favourable prognosis. MRI is needed to confirm presence of isolated ACC after ultrasonographic prenatal diagnosis, as up to 31.4% may have additional anomalies which can significantly impact outcomes. More studies with long term follow up are needed to clarify the true prognosis in isolated ACC.

Audience Take Away:

- The audience will gain awareness of agenesis of corpus callosum, it's ideal diagnostic methods and developmental outcomes
- The audience will gain understanding of the importance in documenting conditions of complete agenesis of the corpus callosum such that future research is aided with more cases in the literature
- The presentation will provide new information on developmental outcomes and pre-natal diagnosis of agenesis of corpus callosum
- Awareness of developmental outcomes will help guide the audiences' approach to prenatal counselling

Biography

Fareha Khalil is a final year medical student at the University of Limerick, Ireland. She has a keen interest in neurological disease, and research in developmental disorders of the brain.

VIRTUAL

Heeya Shah, Omer Usman, Habib Ur Rehman, Sharan Jhaveri, Chaithanya Avanthika, Kamran Hussain, Hamza Islam*, Sailesh I.S.K

Punjab Medical College, Faisalabad, Pakistan

Deep brain stimulation in treatment of parkinson's disease

Parkinson's Disease (PD) is a common progressive neurodegenerative movement disorder. The cardinal feature of Parkinson's is neuronal degeneration causing a dopamine deficit in the brain. This causes a host of clinical features in the patient, however specific clinical criteria for diagnosis are yet to be established. Parkinson's does not have a cure, but a variety of diagnostic and treatment protocols have been developed over the years, with management primarily focusing on pharmacological therapy. Anti-Parkinsonian drugs such as Levodopa lose their efficacy over time and are needed in higher doses as the disease inevitably progresses. An alternative to pharmacological therapy is Deep Brain Stimulation (DBS).

Deep brain stimulation involves transcranial placement of unilateral or bilateral leads (wires) in the subthalamic nucleus or the globus pallidus interna of the brain by stereotactic surgery. Given the multiple hypotheses explaining the different effects of DBS with sometimes conflicting mechanisms, it is difficult to pinpoint the exact way in which DBS operates. Nevertheless, it has proven to be significantly effective. Patients undergoing DBS still need DRT but the dosage is significantly reduced. DBS, although being a cost-effective treatment measure for Parkinson's patients, is not without limitations. A careful selection of patients is required preoperatively that determines the response and tolerance to the therapy in patients.

This review aims to summarize the current literature on DBS in Parkinson's, with a focus on the hypothesized mechanisms, selection criteria, advantages and its limitations.

Audience Take Away:

- Mechanism of DBS
- Diagnosis and Treatment Protocols of Parkinson's
- Role of DBS
- Cost Effectiveness and Comparison of DBS to Standard Treatment Protocols
- Risks and Limitations of DBS

Biography

Hamza Islam is a graduate of Punjab Medical College, Faisalabad, Pakistan. He is an enthusiastic researcher. Internal medicine is among his fields of interest. He was involved in the poster presentations and oral case presentation (CPC) in his Alma-mater. He is interested in presenting his contribution at national and international level. He is working with his fellow researchers on different projects. He also attended multiple health related conferences and workshops. He has published more than 7 research articles in Pubmed indexed journals.

VIRTUAL

Hamza Islam, Sri Madhurima Puttagunta, Rabia Islam*, Sumana Kundu, Surajkumar B. Jha, Ana P. Rivera, Gabriela Vanessa Flores Monar, Ibrahim Sange

Punjab Medical College, Faisalabad, Pakistan

Risk of stroke with mitral stenosis: The underlying mechanism, treatment, and prevention

Mitral stenosis (MS), a valvular heart disease, is defined by the narrowing of the mitral valve orifice. The common risk factors for stroke include Mitral Annular Calcification (MAC), Diabetes Mellitus (DM), male gender, Hypertension (HTN), hyperlipidemia, and obesity. Endothelial damage, hypercoagulability, and blood stasis in the left atrium promote the development of the thrombus. Among all the risk factors described, MAC is the independent predictor of stroke. The complicated mechanisms responsible for thromboembolism, predisposing factors for thromboembolism, the risk of cerebrovascular accident (CVA) in MS patients, advanced standardized assessment models for identifying those at risk for stroke, and the possible advantages and disadvantages of available therapies have all been discussed in this review article. We have also discussed Newer Oral Anticoagulants (NOACs) like dabigatran, edoxaban, apixaban, and rivaroxaban. Non-pharmacological therapies are also highlighted such as left atrial appendage ligation and occlusion devices. We also conducted a thorough review of the literature on the efficacy and safety of various NOACs in reducing the risk of stroke.

Audience Take Away:

- We believe this study can help overcome the challenges by taking a comprehensive view of the correlation between the two elements (MS and stroke) and highlighting the pathogenesis, contributing factors, and management options.
- The risk can be calculated using the CHA2DS2-VASC scoring system.
- Summing up, treatment options for MS include VKAs such as warfarin and NOACs and surgical treatment.
- Successful stroke prevention treatment remains a challenge in high-risk
- patients, necessitating additional evidence from future research.

Biography

I am Dr Rabia Islam, graduated from punjab medical college Faisalabad Pakistan. I have been involved in many poster presentations and oral case presentation projects in my alma matter. I gained years of experience by working under multiple physicians as an Intern and Hospital Physician. I also volunteerd positions at medical facilities in Pakistan and attended multiple health related conferences and workshops. I have remained an active member of Emed (Environment and medicine organization), and took part in multiple health care activities.

VIRTUAL

Mohammad Safdari[1], Zohre Safdari[2], Masoud Pishjoo[3], Sirous Seifirad[4], Daniel Kheradmand[3], Sajjad Saghebdoust*[5]

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[5]Department of Neurosurgery, Razavi Hospital, Mashhad, Iran

Radiological outcome of operative treatment with posterior approach in patients with thoracolumbar junction traumatic injuries: A single-center pilot study in a developing country

Introduction: The thoracolumbar junction represents a transition zone of the spine that leads to a high incidence of fractures. The treatment of burst fractures remains controversial regarding the ideal management. This study assessed the postoperative radiological outcome of Thoracolumbar Junction (TLJ) fixation in patients with TLJ injuries who underwent surgery.

Material and methods: All traumatic patients with TLJ injuries who were referred to the Khatam hospital of Zahedan between 2015-2020, with their Thoracolumbar Injury Classification And Severity Score (TLICS) of four or more and who underwent surgery, were included in this study. The patients who entered the study were called for a follow-up examination. The degree of kyphosis, Proximal Junctional Kyphosis (PJK), and fusion were assessed in these patients.

Results: Among 273 patients, the average age was 43.5±12.3 (21-73) years. One hundred ninety-eight patients (72.5%) had no neurological symptoms at admission. Based on the above criteria, the kyphosis angle of these patients was calculated before surgery, which in 46 patients (16.8%), the kyphosis angle was more than 25 degrees. Pre-operation kyphosis was significantly associated with follow-up kyphosis ($p < 0.001$). Evidence of no fusion was also observed in 22 patients (8.1%). According to the Chi-Square test, no association was observed between preoperative kyphosis and postoperative complications, including PJK and fusion ($p > 0.05$).

Conclusions: According to our study, the posterior spinal fixation procedure is a low-complication method with an acceptable radiological outcome. Although kyphosis before surgery is a factor in developing long-term kyphosis, it is not associated with nonfusion and PJK.

Audience Take Away:

- Most spinal fractures are stable, do not require surgery, and are often cured with proper braces or hyperextension. The benefits of spinal fracture surgery include better correction of kyphotic deformity, greater stability, the possibility of direct and indirect decompression of neural elements, less need to use external immobilization, and faster return to work.
- According to previous studies, surgical indications based on the presence of canal compromise, neurological defects, reduction in body height, and the presence of kyphosis were used relatively. In a study by Reid et al., the researchers concluded that patients harboring burst fracture with neurological defect or kyphosis angle greater than 35 degrees had to undergo surgery.
- In 2005, Vacarro et al. introduced a new classification for thoracolumbar fractures called thoracolumbar injury classification and severity score (TLICS). According to this classification, patients who receive a score of 3 or less do not need surgery; however, patients with a score of 5 or higher must undergo surgery. In cases where the patient's score is 4, the type of treatment is based on the surgeon's judgment.

- Krompinger et al. found that about 36 percent of burst fractures in their follow-up examinations had changed more than 10 degrees. This study also showed that a significant portion of patients has an increase in kyphosis during follow-up examinations, which highlights the importance of examining patients in the long term. Siebenga et al. showed that regardless of the clinical consequences, surgery is more cost-effective than conservative treatment in the burst fractures TLJ
- Based on the results of previous studies, in TLJ traumatic injuries, conservative treatment is associated with significant complications such as prolonged immobility, delayed return to work, higher chances of developing kyphosis, and less spinal stabilization.
- According to the general results and radiological studies of follow-up in our study, it can be concluded that the posterior spinal fixation procedure is a low-complication method and with an acceptable radiological outcome. Although kyphosis before surgery is a factor in developing long-term kyphosis, it is not associated with nonfusion and PJK.

Biography

Dr. Sajjad Saghebdoust studied medicine at Mashhad University of Medical Sciences and attained 8th place in the national pre-internship examination, thereby becoming a member of Iran's National Elites Foundation. He graduated as MD in 2021 and obtained MBA and MPH degrees thereafter. Meanwhile, he was awarded two neurosurgery research positions in two well-known neurosurgical centers, the Neurosurgery Department of Razavi Hospital (Mashhad, Iran) and the Sports Medicine Research Center at Tehran University of Medical Sciences. Since then, he has been involved in a wealth of cutting-edge neurosurgical research projects under the supervision of several distinguished neurosurgeons.

VIRTUAL

Sajjad Saghebdoust*[1], Seyed Ghavam Shafagh[2], Neda Pak[3], Reza Fekrazad[4], Masoud Khadivi[5], Morteza Faghih Jouibari[5], Mohammad Reza Boustani[5,6]

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Role of percutaneous laser disc decompression in patients with lumbar disc herniation on pain relief: A quasi-experimental pilot study

Introduction: Disc herniation is broadly defined as a localized or focal displacement of disc material beyond the limits of the intervertebral disc space. The disc material may be the nucleus, cartilage, fragmented apophyseal bone, annular tissue, or any combination thereof. Laser surgery is one of the treatment modalities for treating patients with lumbar disc herniation. This study aims to examine the effect of Percutaneous Laser Disc Decompression (PLDD) in patients with lumbar disc herniation.

Material and methods: This study was conducted on 58 patients who underwent PLDD (optical fiber inserted through an 18G needle, 8 joules, and 8 watts). Baseline characteristics of patients include age, gender, and Body Mass Index (BMI) were recorded. The pain was assessed through a 10-point Visual Analogue Scale (VAS) with endpoint anchors of no pain (0 points) and severe pain (10 points) before and after the procedure. Also, we compared the VAS based on age, gender, and BMI.

Results: The study population consisted of 58 patients, among which 58.6% were male. The mean age of the participants was 63.19±13.48 years. The mean BMI of the participants was 29.09±6.51 Kg/m2. The participants suffered from symptoms associated with disc herniation for an average of 30.44±5.01 weeks, and 55.2% had symptoms for more than 30 weeks. The mean VAS score before and after surgery was 8.73±1.29 and 5.22±2.7, respectively, which showed a marked reduction ($P < 0.001$). We found no significant association between VAS and gender, age, BMI, and symptom duration when assessing the contributing factors to the patients' pain level.

Conclusions: Our study showed that using PLDD for carefully selected individuals can reduce pain and disability in patients as a safe, non-invasive procedure.

Audience Take Away:

- PLDD's current indications are radicular pain, lumbar spinal stenosis, and disc herniation. PLDD has been shown to improve the clinical outcome of contained lumbar herniated discs.
- The PLDD patient selection criteria are described in the literature as leg pain is worse than LBP, herniated disc on MRI, chronic LBP lasting more than three months, non-invasive treatment failure, no evidence of neurological deficiency, segmental instability, preservation of more than 75% of the disc height, and no sign of psychogenic component
- In previous studies, it has been shown that the effects of PLDD in selected patients reported a 70% success rate over a 5-year follow-up period with no complications.
- The patients' post-PLDD pain can be reduced. Hence, PLDD can be considered an appropriate method for treating lumbar disc herniation in carefully selected patients

Biography

Dr. Sajjad Saghebdoust studied medicine at Mashhad University of Medical Sciences and attained 8th place in the national pre-internship examination, thereby becoming a member of Iran's National Elites Foundation. He graduated as MD in 2021 and obtained MBA and MPH degrees thereafter. Meanwhile, he was awarded two neurosurgery research positions in two well-known neurosurgical centers, the Neurosurgery Department of Razavi Hospital (Mashhad, Iran) and the Sports Medicine Research Center at Tehran University of Medical Sciences. Since then, he has been involved in a wealth of cutting-edge neurosurgical research projects under the supervision of several distinguished neurosurgeons.

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Patel Shreenal*, Srinivasan Aswin, Patel Keya, Tatineni Lakshmi, Rizvi Khulood
HCA Healthcare Houston, Kingwood, United States of America

A rare case of probable neurosarcoidosis

Introduction: Neurosarcoidosis is an uncommon manifestation and should be suspected in any patients with sarcoidosis with new neurological symptoms. We present a rare case of probable neurosarcoidosis, which was diagnosed by exclusion and elevated Acetylcholinesterase (ACE) levels in the CSF.

Case presentation: A 62-year-old female with a history of HTN, DM, CHF, sarcoidosis, lung cancer (in remission), presented to the Emergency Department (ED) for seizure and encephalopathy. Vitals were unremarkable except for temperature of 100F and tachycardia 155 beats/min. Initial labs were unremarkable except for lactic acidosis 2.7 and Na 132. CT and MRI brain were unremarkable. Lumbar puncture was significant for WBC 1643 with 96% PMNs, glucose 51 and TP 153. We began treatment for bacterial meningitis with broad spectrum antibiotics, however all the subsequent testing for bacterial/viral/fungal suspects were negative. She also tested negative for RPR, HSV and HIV. The patient had to be upgraded to the ICU briefly as there were concerns that she was not able to protect her airway. It was discovered here that she was supposed to be on prednisone at home for sarcoidosis but non-compliance likely played a role. Her home dose of prednisone was resumed and after that, the patient began to improve drastically and she was discharged to a skilled nursing facility. The patient presented back to the ED three weeks later for encephalopathy and right sided hemiparesis. Initial labs and vitals on this presentation were unremarkable. MRI brain showed a subtle diffusion weighted signal and flair hyperintensity within the left parietal, left temporal and left occipital lobes. She underwent another lumbar puncture, which was remarkable for an elevated ACE level. Given the MRI and CSF findings, we made a diagnosis of probable neurosarcoidosis and initiated high dose steroids. She began to improve with the addition of steroids and was eventually discharged back to a skilled nursing facility with a steroid taper.

Discussion: Neurosarcoidis is a rare form of sarcoidosis in which inflammation is deposited along any part of the CNS. Symptoms are dependent on the location of the inflammation. In our patient, these symptoms manifested as seizures, hemiparesis and encephalopathy. Biopsy is the gold standard of diagnosis, however obtaining one can be challenging. Therefore, many cases are diagnosed as probable neurosarcoidosis. To make this diagnosis, there needs to be high clinical suspicion, MRI/CSF findings suggestive of neurosarcoidosis and exclusion of all other pathologies. CSF ACE may be another tool to aid in the diagnosis of neurosarcoidosis. Although there is no consensus, there have been several studies done which indicate a strong correlation between elevated CSF ACE levels and neurosarcoidosis.

Conclusion: The gold standard for the diagnosis of neurosarcoidosis is a tissue biopsy which may be difficult to achieve. Therefore, neurosarcoidosis is a diagnosis of exclusion after all other pathologies have been ruled out.

Audience Take Away:

- This abstract highlights the importance of keeping neurosarcoidosis in the differential whenever a patient with known sarcoidosis presents with neurological symptoms
- In the clinical setting, biopsy may be difficult to obtain to diagnose this condition; CSF ACE may be a helpful tool in such cases
- This case also highlights the importance of compliance to steroids for those diagnosed with sarcoidosis

Biography

Dr. Shreenal Patel completed her bachelors of science in biology at the University of Georgia in 2015. She worked as a medical scribe in Atlanta, Georgia from 2015 - 2016 before attending medical school at William Carey College of Osteopathic medicine from 2016 - 2020. Currently, she is a second year internal medicine resident at HCA Healthcare Houston.

VIRTUAL

Hannah Girgis*, John Clarke, Michael Mader
Department of Radiology, Aultman Hospital, Canton, OH, United States

A de novo case of neurofibromatosis type 2

NeuroFibromatosis type 2 (NF2) is a genetic disorder that predisposes individuals to multiple tumors of the nervous system. The most common of these are bilateral vestibular schwannomas; intracranial and spinal meningiomas, and spinal cord ependymomas. The condition is caused by mutations in the NF2 tumor suppressor gene MERLIN located on chromosome 22.The mutations are inherited in an autosomal dominant fashion in approximately 50% of patients and arise de novo in the other 50%. Up to 25-30% of individuals with NF2, either inherited or de novo, have mosaicism. These mosaic individuals only carry the mutation in some of their body cells, and therefore often develop less severe symptoms. In like manner, in a patient with mosaic NF2 there is less chance of passing on the condition whereas children of non-mosaic NF2 patients have a 50% chance inheriting the mutation.

MRI with special attention to the internal auditory canals revealed an avidly and uniformly enhancing lesion within the right Internal Auditory Canal (IAC). The lesion caused mild expansion of the right IAC and extended into the right cerebropontine angle cistern. Imaging characteristics most compatible with a vestibular schwannoma.

Neurofibromatosis type 2 (NF2) is an inherited disease with a prevalence of 1 in 60,000 and birth incidence of 1 in 25-33,000 individuals3. The disease can present with many neurologic findings including bilateral vestibular schwannomas, central nervous system meningiomas, as seen in this patient, peripheral neuropathy as well as others. Many individuals begin having hearing loss and balance problems, on account of the vestibular schwannomas. These symptoms often account for the earliest signs of the disease. Continued research on treatment and management has developed increased treatment options for individuals diagnosed with NF2. These include neurosurgery, radiosurgery, auditory rehabilitation, and molecular biology3. Unfortunately, with the rarity of NF2, research is often limited, and randomized control trials are difficult to take forward.

When clinically evaluating a patient with suspected NF2, a thorough neurological evaluation, including visual examination, as well as hearing and gait assessment. The predominant issues found on physical evaluation are often impaired balance and issues with social communication3. Further assessing with imaging, often with contrast-enhanced T1-weighted MRI, is commonly indicated and family members with known mutations are screened and followed with MRI.

Audience Take Away:

- Description of Neurofibromatosis Type 2 and Incidence Statistics that will allow guidance and assistance in diagnosis
- Characteristics of patients presenting with Neurofibromatosis Type 2 to increase awareness of the disease and diagnosis
- Pathognomonic Imaging Findings for Neurofibromatosis Type 2 to help physicians recognize these findings on radiological imaging
- General Neurological Findings for Neurofibromatosis Type 2

Biography

Hannah Girgis studied Chemistry at Youngstown State University, United States and graduated with a Bachelor's in Science after two years in 2019. She then began medical school the same year and has been working towards her M.D. and Master's in Medical Ethics and Humanities (MEH) since then.

Joshua Burshtein*[1], Aaron Burshtein[1], Reuben Burshtein[2]

[1]Department of Neurology, Donald and Barbara Zucker School of Medicine at Hofstra / Northwell, Hempstead, NY, USA
[2]Department of Neurology, Catholic Health Services, West Islip, NY

Cost analysis of inpatient bacterial meningitis hospitalizations

Background & Purpose: Knowledge surrounding inpatient costs of Bacterial Meningitis (BM) management is limited. We aimed to explore the financial burden of hospitalization due to BM, including cost trends over time and factors associated with higher costs.

Methods: We performed a cross-sectional, retrospective analysis of BM hospitalizations in a publicly available database, Washington state comprehensive hospital abstract reporting system, from 2014-2020. Costs were evaluated based on parameters including demographics, type of admission, Length of Stay (LOS), and discharge destination. Statistical analysis was performed using SPSS version 28.0.

Results: From 2014-2020, 292 patients were hospitalized for BM. Majority of admissions were age 18-64 (63%), followed by 0-17 (23%) and ≥65 (14%). Most were admitted emergently vs. urgently (70.2 vs. 26.4%), and during the weekday (72%). LOS 0-7 days (61%) was most prevalent. Inpatient mortality rate was 4.45%. Aggregated hospitalization cost was $26,161,000. Mean cost per hospitalization was $89,592.47 (SD=$100,988.25), with no difference in cost per year (p=0.486). The mean rate of BM hospitalization was 6 per 100,000 patients. Those age 0-17 had higher mean cost than age 18-64 ($123,328.36 vs. $79,114.75, p=0.006) and age 65+ ($123,328.36 vs. $81,428.57, p=0.1). As LOS increased from 0-7 days to 8-14 days to 30+ days, cost increased (p<0.001). Urgent visits had $43,029.33 higher cost than emergent visits (p=0.004). Linear regression showed LOS 8-14 days, 15-30 days, 30+ days each predicted higher cost vs. LOS 0-7 days (p<0.001, p<0.001, p<0.001, respectively). Urgent visits predicted higher cost vs. elective visits (p=0.002). Expired patients predicted a cost of $85,644.95 greater than those discharged home (p<0.001).

Conclusion: These findings demonstrate BM hospitalization cost continues to have substantial economic burden on patients. Cost has been stable over this 7-year period. Younger age, greater LOS, and urgent visits had higher hospitalization cost. LOS, urgent visits, and expired patients predicted higher cost.

Audience Take Away:

- With the rising cost of healthcare, it is vital to understand the current cost burden of bacterial meningitis for patients. This study provides the most recent cost analysis for hospitalization of bacterial meningitis in the USA
- Physicians and researchers can use this data to understand the patient populations they are treating, and therefore explore methods of decreasing overall costs. This may lead to research into efficient use of diagnostic techniques, biomarkers ordered, and treatments and other prescribed interventions

Biography

Dr. Burshtein studied Biology at New York University, USA and graduated as BA in 2018. He then completed medical school in 2022 at the Donald and Barbara Zucker School of Medicine at Hofstra/Northwell in New Hyde Park, NY. Upon graduating, he will be starting a preliminary year in the Internal Medicine Residency Program at The Icahn School of Medicine Mount Sinai Morningside/West in New York. He has conducted clinical research for the past 8 years in various medical specialties, has been published numerous times in medical journals, and has presented at regional and national medical conferences.

VIRTUAL

David Galel*[1], Joseph Reynolds[2], David Everly[2]

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Multiple sclerosis disease modifying pharmacotherapies effect on EBV infection

Multiple Sclerosis (MS) is an autoimmune disease targeting myelin in the central nervous system. Most Disease Modifying Therapies (DMTs) for MS function via immunomodulation by a variety of modalities, some of which are immunosuppressive and some which are not. In this study we compare various DMT regimens to metrics of Epstein-Barr Virus (EBV) infection.

Fifty-nine MS patients participated, of which 46 were on DMT regimens of aubagio, avonex, betaseron, copaxone, gilenya, mayzent, plegridy, rebif, tecfidera, or tysabri. Participants on DMT regimens were categorized by immunosuppressive vs non-immunosuppressive therapies per guidance from a regional neurology physicians' group, with immunosuppressive DMTs including avonex, betaseron, copaxone, plegridy, and rebif. Fifty-two volunteers without MS served as controls. Peripheral blood was collected from each participant in a red-top clot tube to isolate serum. A portion of serum was analyzed by quantitative PCR. Results were reported as qualitative incidence of infection (number of participants with >0 viral copies), and quantitatively as the mean copy number for participants with detectable viral copies. Another portion of serum was incubated with a microarray chip containing 110 unique EBV peptide epitopes. Secondary anti-IgG fluorescently conjugated antibodies were added, and the resulting chips were analyzed for fluorescence to determine IgG reactivity to each EBV epitope. ANOVA and T-tests were used to compare metrics between the groups with significance determined at $p<0.05$.

Rates of infection in control, MS without DMT, MS with immunosuppressive DMT, and MS with non-immunosuppressive DMT groups were 51%, 50%, 8%, and 10% respectively. For those with detectable copies the mean ± standard deviation copy numbers were 57.30 ± 46.24, 61.80 ± 62.72, 22.75 ± 7.28, and 26.60 ± 13.58 respectively, with ANOVA $p=0.6261$. IgG antibody reactivity per 110 epitopes were 35.52 ± 20.81, 38.38 ± 14.48, 42.13 ± 14.71, and 36.25 ± 15.62 respectively, with ANOVA $p=0.6473$. T-testing between individual groups did not yield significance for mean viral copy numbers nor antibody reactivity.

Rates of EBV infection were notably higher in the control and MS without DMT groups which contradicts the notion that participants on immunomodulating DMTs might have greater susceptibility to infection. However, our study did not yield significant differences between MS participants with different DMT regimens or the control group in terms of EBV viral burden or antibody reactivity. This suggests that the DMT regimens assessed here do not result in increased susceptibility to EBV. Differences in EBV infection rates are a surprising result that warrants further investigation.

Audience Take Away:

- This presentation informs the audience about the effects of disease modifying pharmacotherapies on the susceptibility of multiple sclerosis patients to viral infections
- Audience members will gain an understanding of how viral burden of Epstein Barr Virus compares between MS patients with immunosuppressive pharmacotherapies, non-immunosuppressive pharmacotherapies, and patients without MS-related pharmacotherapies

- Audience members will observe the impact of various MS-related pharmacotherapies on Epstein Barr Virus burden and antibody reactivity, which may inform decision making for pharmacotherapy intervention or specific drug choice

Biography

David Galel studied Human Biology at the University of California, San Diego and graduated with BS in 2014. He then worked at the Cellular Therapy laboratory at the San Diego Blood Bank where he conducted research in predictive metrics of stem cell content in umbilical cord blood. He is now an MD candidate at the Chicago Medical School where he is researching the role of infection by common viruses in the pathogenesis of multiple sclerosis.

VIRTUAL

Neil Gerts*[1], Trevor Owens[2], Preetivi Ellis[3], Venkat Aachi[4]

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Covid infection and the surprise guillain barre syndrome diagnosis

The World Health Organization declared COVID-19 a pandemic in February 2020, and it has affected the life of each and everyone. COVID-19 is designated as severe respiratory syndrome coronavirus 2 (SARS-CoV-2). It causes an acute inflammatory process in the body, which is hypothesized to be the basis of Guillain Barre Syndrome (GBS), which is often triggered when an immune response to an antecedent infection or another event cross-reacts with the shared epitopes on peripheral nerves in a process called molecular mimicry. Classic symptoms included ascending paralysis, blurry vision, and weakness. Diagnosis is by lumbar puncture which will demonstrate albuminocytological dissociation, and treatment is by Intravenous Immune Globulin therapy (IVIG). Here we present a case of Guillain Barre Syndrome in an adult who was recently infected with COVID-19 and still in the active phase of the infection. The patient originally presented with complete lower extremity paralysis and tested positive for COVID-19 on admission. As an MRI of the full spine was negative, the patient underwent a lumbar puncture which was suggestive of Guillain Barre Syndrome. After a five-day course of IVIG, the patient had complete resolution of his symptoms.

Audience Take Away:

- As new information is being discovered about COVID-19 every day, we hope to demonstrate the association between COVID-19 and GBS
- We hope to help medical professionals recognize the clinical presentation of GBS in a patient with an active COVD-19 infection, and the workup, management, and treatment associated with GBS
- We hope to familiarize the audience regarding the association between COVID-19 and GBS, and aid in the early recognition and diagnostic approach regarding GBS

Biography

Dr. Neil Gerts received his Doctor of Medicine at St. George's University School of Medicine, Grenada in 2021. He is currently in his first year of his Internal Medicine Residency at Los Robles Regional Medical Center in Thousand Oaks, CA.

Rebecca Simon*, Jason Morris, Steven Vassil, Khloe Gu, Jessica Okun
Department of Health Professions, Nova Southeastern University Dr. Kiran C. Patel College of Osteopathic Medicine, Fort Lauderdale, FL, USA

Intracranial mass: Meningioma vs. thrombosed aneurysm

Correct identification and analysis of intracranial masses is of high clinical importance especially when it comes to treating acute illnesses and focal neurological deficits, while preventing further deterioration.

We present a rare case of a giant thrombosed aneurysm in the intracavernous segment of the right Internal Carotid Artery (ICA). The patient, a 75-year-old female, presented with nine months of progressive headache, right cranial nerve III palsy, and retroorbital pain with no significant past medical history. The initial CT (computed tomography) scan of the brain revealed a suprasellar and right periclinoid calcified mass, forming an initial suspicion for a meningioma. Further imaging consisted of a CT angiogram of the head and neck, which showed occlusion in the cervical and clinoid region of the right ICA with preserved right ACA (Anterior Cerebral Artery) and MCA (Middle Cerebral Artery) blood flow via collateral circulation.

Following a neurosurgery consult for the diagnosis of a meningioma, the patient was placed on lovenox for DVT (Deep Vein Thrombosis) prophylaxis. A subsequent IR (Interventional Radiologic) cerebral angiogram obtained four days later revealed recanalization of the previously occluded right ICA with a large dissecting aneurysm in the petrous/cavernous segment. This revelation immediately redirected the treatment plan for this patient, as neurosurgeons were now dealing with an aneurysm as opposed to a meningioma. This case describes a perplexing situation that led many physicians to believe this was a meningioma, based on radiographic findings. However, administration of lovenox led to a completely different diagnosis. The aneurysms was previously completely thrombosed and angiographically occult in the initial CT scan, leading to the diagnosis of a meningioma. When the patient received lovenox, this caused the aneurysm to rebleed and the patient to become symptomatic. The CTA confirmed this as the aneurysm had recanalized as a result. Therefore, administering Lovenox can drastically change the features of an angiographically occult thrombosed aneurysm and yield diagnostically significant clues on the genesis of focal neurological deficits.

Premature resection of a suspected meningioma prior to further radiographic investigation would have put the patient at risk for perioperative aneurysm rupture, resulting in massive intracranial hemorrhage and other life-threatening complications. The aneurysm was ultimately treated with Pipeline Flow Diverter Embolization (PFDE) and the patient is clinically stable as of today. This case reflects the critical importance of considering differential diagnoses when a sphenoid mass is discovered and it does not appear to homogeneously enhance as a typical meningioma would.

Audience Take Away:

- This will assist healthcare professionals that are faced with similar presentations in expanding their differential diagnoses for intracranial masses
- Radiographic identification of intracranial masses and understanding that aneurysms may appear differently when thrombosed vs. when it is recanalized and blood flow is restored
- Keeping in mind the consequences of operating on intracranial masses that my result in active bleeding

- Understanding the consequences of using anticoagulants and how it can change interpretation of intracranial radiographic findings
- Utility of the pipeline diverter flow embolization device in the treatment of intracranial aneurysms

Biography

Rebecca Simon OMS-III is a medical student at Nova Southeastern University and is planning to pursue a neurology residency. She received her BS in Neuroscience & Behavior from Florida Atlantic University in 2018 and her MS in Physiology from Georgetown University in 2019, where she completed research in epilepsy.

Mohammad Abu-Abaa
Capital Health Regional Medical Center, Internal Medicine Residency Program, Trenton, NJ, USA

How uncommon presentation of hyperthyroidism culminated in wernicke's encephalopathy

A 56 years old female patient presented with a few weeks of persistent nausea, vomiting, watery diarrhea, generalized itching and unintentional weight loss. She denied alcohol intake. Labs showed mild hypercalcemia at 10.6 mg/dl. Abdominal examination was benign. Investigations revealed hypercalcemia with pancreatitis. Thyroiditis was also evident with positive thyroid stimulating antibodies. Her pancreatitis was managed conservatively and was presumed to be biliary origin, and a cholecystectomy was pursued. Methimazole at low dose was started after surgery. Persistent nausea and vomiting prompted Esophagogastroduodenoscopy (EGD) that was unremarkable. Physical exam showed an erythematous beefy tongue suggestive of glossitis. Vitamin B1 and B6 levels were low. Video fluoroscopy showed oropharyngeal dysphagia. Neurological exam showed diffuse □ weakness, decreased recent and remote memory with poor orientation. CT head was unremarkable. 24 hour electroencephalogram (EEG) was unyielding. Magnetic resonance imaging of the brain showed FLAIR hyperintensity at central pons and bilateral thalami. No disturbances of sodium level was evident. Her mental status continued to worsen rapidly within a few days and she became minimally responsive, hypothermic and hypotensive and as such she was intubated for airway protection. Cerebrospinal fluid analysis was unremarkable. Given the possibility of autoimmune encephalitis, she received empiric plasmapheresis and steroids. Lack of improvement prompted a consideration of thiamine replacement. Repeat MRI after a few days showed improving thalamic hyperintensities with improvement in mentation. Extensive workup was unyielding for malignancy, syphilis, Human Immunodeficiency Virus (HIV), Systemic Lupus Erythematosus (SLE), Sjogren syndrome, scleroderma, paraneoplastic and autoimmune encephalitis panels. Extubation was feasible after placement of tracheostomy and percutaneous endoscopic gastrostomy. Over the course of over one month, improvement was evident as she was able to speak with partial regain of muscle power with daily thiamine supplement.

This case serves to remind clinicians of the uncommon link between hyperthyroidism and non alcoholic Wernicke's Encephalopathy (WE). Intractable vomiting and poor oral intake can be the main presentation of hyperthyroidism. This, along with hypermetabolic state in hyperthyroidism, can induce rapid thiamine depletion. It also reminds us of atypical manifestations of WE.

Audience Take Away:

- Mechanism of WE in hyperthyroidism
- Risk factors for non alcoholic WE
- Typical and atypical MRI findings in WE

Biography

Dr. Abu-Abaa graduated from the University Of Basrah College Of Medicine in Iraq in the top 5% of his class. Following graduation, he completed a Transitional Year of training followed by two years of Internal Medicine and six months of neurology training experiences. Dr Abu-Abaa also volunteered in underserved areas with the Iraqi Ministry of Health. He is currently PGY2 at Capital Health Regional Medical Center Internal Medicine residency program.

VIRTUAL

Patrick Brown*[1], Jose Posas[2]

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[2]Ochsner Neurology Residency Program Director, Sports Neurology, Ochsner Health, New Orleans, LA, USA

A system-wide retrospective cohort analysis of the development of psychiatric conditions following concussion

Objective: The purpose of this study was to evaluate the development of clinically diagnosed anxiety or depression within one year following a minor Traumatic Brain Injury (mTBI).

Design: A state-wide hospital system (Ochsner Health) retrospective cohort analysis

Setting: This study investigates the development of clinically diagnosed anxiety and/or depression within a set one-year time period following mTBI.

Participants: 2,960 patients presenting to the ochsner health system between 2010 and 2020 with clinically diagnosed mTBI and developed a psychiatric condition (including all mental, behavioral and Eurodevelopmental disorders). Patients had no psychiatric conditions prior to mTBI.

Out-come measures: Development of anxiety and/or depression, according to ICD-10 criteria.

Main Results: Of the 2,960 mTBI patients, 90 (3.04%) developed diagnosed anxiety alone, 60 (2.02%) developed clinical depression alone, and 112 (3.78%) patients developed both anxiety and depression within one year of mTBI.

Conclusions: Our findings provide further evidence of the development of psychiatric conditions within one year of mTBI, and outline the importance of the psychiatric aspect of brain injuries.

Audience Take Away:

- This study provides insight into the psychiatric risks following concussion injuries that physicians must be aware of
- This study provides proof for the development of anxiety and depression within one year of minor traumatic brain injury, further supporting evidence from prior studies
- This study indicates the important of a psychiatric workup following brain injury, and caution when treating only the physical symptoms of concussion

Biography

Patrick Brown is a 4th year medical student at the University of Queensland – Ochsner School of Medicine. He studied Biology at California Polytechnic State University in San Luis Obispo, California, before going to medical school. He has spent his first two years studying medicine at the University of Queensland in Brisbane, Australia, until moving to New Orleans, Louisiana to complete the second half of his unique four year program. He has a keen interest in Neurology and hopes to contribute to the diverse research surrounding the field.

VIRTUAL

Dominic Bruegger[1], Anna Lucia Koth[1,2], Anthia Papazoglou*[3], Muriel Dysli[1,3], David Goldblum[4], Mathias Abegg[1], Markus Tschopp[1,3], Christoph Tappeiner[4]

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[2]Augenpraxis Untertor, Winterthur, Switzerland
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[4]Pallas Kliniken, Olten, Switzerland

Evaluation of the reddesa chart, a new red desaturation testing method, for optic neuritis screening and grading in clinical routine

Background: Optic neuritis and optic neuropathy can cause a reduced color-sensitivity. Therefore, in clinical routine the patient's perception of the red color is tested in order to evaluate the function of the optic nerve. Red desaturation is traditionally tested by presenting an object of red color to the patient (red cup test). The purpose of this study was to evaluate the potential of a new red desaturation test based on polarization we created, the reddesa chart, as a screening method for optic neuropathy.

Methods: For this monocentric prospective pilot study, 20 patients with unilateral optic neuritis and an equal number of healthy controls were included. In all participants the Best Corrected Visual Acuity (BCVA) was assessed, slit lamp examination of the anterior and posterior eye segments was performed, and the Relative Afferent Pupillary Defect (RAPD) and the red desaturation with the red cup test and the Reddesa chart was tested.

Results: The mean BCVA in the optic neuritis group was 0.76±0.36 in the affected eye (95% of eyes with RAPD, 75% of eyes with difference in the Reddesa test) and 1.28±0.24 in the healthy eye, while in the control group BCVA was 1.14±0.11 in the right eye and 1.15±0.14 in the left eye (none of the eyes with RAPD or abnormal Reddesa test). The Reddesa chart test demonstrated a positive predictive value of 100% and a negative predictive value of 80% for detecting optic neuritis.

Conclusion: The reddesa chart is a useful tool to more objectively test and possibly quantify red desaturation in clinical routine.

Audience Take Away:

- An easy and practical solution to test red desaturation by all kind of medical personnel and not only by ophthalmologists or neurologists
- A more objective way to test for red desaturation
- A potential way to quantify red desaturation

Biography

Dr. Papazoglou studied Human Medicine at the Kapodistrian University of Athens, Greece and graduated with honors in 2011. She conducted a Master of Science in Ophthalmic Imaging at the Democritus University of Thrace, Greece, while completing her ophthalmology residency and her medical dissertation at the University Hospital of Zurich. She joined her current Hospital, where she completed her ophthalmosurgical subspecialty in 2018.

Pourya Bahiraei*[1], Aysan Jalili[2]

[1]Medical Student, Students' Scientific Research Center, Tehran University of Medical Sciences, Tehran, Iran
[2]Msc Student of Physiology, Department of Animal Biology, Faculty of Biological Sciences, Kharazmi University, Tehran, Iran

Neuropsychiatric and cognitive aspects of behcet's syndrome: A systematic review on recent studies

Background: Behcet's Syndrome (BS) is an idiopathic vasculitis disease, which causes a wide spectrum of different focal and systemic symptoms. This review aimed to investigate the neuropsychiatric and cognitive aspects of this syndrome.

Method: Using the keywords "psychiatric disorders and Behcet's syndrome", "cognitive deficits and Behcet's syndrome" and related sub-categories, We searched among the articles of PubMed, Google Scholar, and Scopus databases. By removing duplicates and using inclusion-exclusion criteria, 35 articles were finally chosen to be reviewed.

Results: Investigating the prevalence of mood disorders, especially depression and bipolar disorder among patients with BS, compared to the control group has monopolized the largest portion of these studies. Their results show a higher frequency of mood disorders in patients with BS compared to the healthy control group. In performing cognitive tests related to measuring memory, learning abilities and decision making, patients with Behcet's syndrome scored lower than the control groups.

In another study, complaints of sleep disorders in patients with BS were significantly more seen than in the control group. Also, the amount of complaints about sexual disorders, in general, is higher among patients with BS compared to control groups. Most of these complaints are erectile dysfunction in males.

Conclusion: A short review of recent studies shows we need more reliable clinical research to redefine Behcet syndrome due to its cognitive and behavioral aspects. However, future studies are also facing many challenges, among which we can point out the wide variety of symptoms, access and long-term follow-up of patients, and the inevitable presence of Confounding bias.

Audience Take Away:

- Despite it seems we know all aspects of Behçet's syndrome very well. But the cognitive and behavioral aspects of Behcet´s are mostly hidden from the attention of clinical experts. This review article is an attempt to learn more about the less seen aspects of this disease
- This study can be helpful for clinical specialists to have a comprehensive look at Behcet´s in the path of diagnosis and also treatment
- Also, this topic can be a spark for researchers in the interdisciplinary fields of neuroimmunology and neurorheumatology

Biography

Pourya Bahiraei is a junior researcher in the field of clinical neuroscience and cognitive neurology.He won a bronze medal in "stem cells and regenrative medicine" national student olympiad (2016). He entered the Tehran Faculty of Medical Sciences by getting the top 1 percent rank in the national university entrance exam)2017(And in 2019, he founded the EHIA Neuroscience Association of Tehran University of Medical Sciences.

Participants List

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We wish to meet you again at our upcoming event

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