**PLENARY**

| **Friday October 2, 2020** | |
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| **09:00-11:00** | **SESSION 1 \| OPENING SESSION** |
| 09:00-09:15 | Welcome remarks<br>**Amos Korczyn,** Israel and **Anthony Schapira**, UK |
| 09:15-09:30 | Welcome from the Association of British Neurologists (ABN)<br>**Tom Warner**, UK |
| 09:30-10:00 | Parkinson's disease: From mitochondria to lysosomes and on to therapy<br>**Anthony Schapira**, UK |
| 10:00-10:30 | Digital health technologies in clinical care and clinical trials |
| 10:30-11:00 | Should cannabinoids be legally available to the public? |
| **11:00-11:30** | *Coffee Break* |
| **11:30-13:00** | **SESSION 2 \| THE MENTAL STATE** |
| 11:30-12:00 | Neurostimulation and its potential to improve learning and cognition in health and disease |
| 12:00-12:30 | Functional neurological disorders<br>**Stoyan Popkirov**, Germany |
| 12:30-13:00 | Pathological impulsivity - a neurobehavioural phenomenon |
| **13:00-13:45** | *Lunch Break* |
| **13:45-15:15** | **SESSION 3 \| NEUROIMMUNOLOGY** |
| 13:45-14:15 | GAD antibodies spectrum disorder |
| 14:15-14:45 | Multiple Sclerosis is one disease; why definitions matter to patients and to how we treat them |
| **15:15-16:45** | **SESSION 4 \| DEMENTIA** |
| 14:45-15:15 | Multimorbidity of the ageing brain<br>**Johannes Attems**, UK |
| 15:15-15:45 | Emerging neuropathological comorbidities in aging - LATE, ARTAG and CTE<br>**Lea Grinberg**, USA/Brazil |
| 15:45-16:15 | The two in one brain circulations |
| 16:15-16:45 | Dementia and the arts<br>**Sebastian Crutch**, UK |
| **16:45-17:00** | *Coffee Break* |
| 17:00-18:00 | **GWAS is likely to contribute significantly to Alzheimer's disease (AD) patients care.**<br>*Capsule: Genome-wide association studies (GWAS) looking at genetic variants in different individuals may identify variants associated with disease. These cross-national investigations have been applied widely to AD, identifying dozens of "disease-specific" alterations. Is the huge and expensive investment likely to help in patient care?* |
| 17:00-17:10 | Host: **Peter Whitehouse**, USA |
| 17:10-17:25 | Yes: |
| 17:25-17:40 | No: **Amos Korczyn**, Israel |
| 17:40-18:00 | Discussions and rebuttals |

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| 18:00-18:30 | Thomas Willis and the first decade of the brain<br>**Alastair Compston**, UK |
| 18:30-19:00 | Welcome to CONy 2020 |
| **19:00** | **Networking Reception** |

**MULTIPLE SCLEROSIS (MS)**

| **Saturday October 3, 2020** | |
|---|---|
| **07:30-08:30** | **E-Poster Presentations (Exhibition Area)** |
| **08:30-10:10** | **DIAGNOSIS** |
| **08:30-09:20** | **Are the 2017 MS McDonald criteria too liberal and should be more restrictive?**<br>*Capsule: The 2017 revisions of the McDonald criteria for the diagnosis of MS were mainly designed to facilitate an earlier MS diagnosis and the clinically isolated syndrome. While the criteria are easy to use and highly sensitive, they lack specificity and may bear the risk of MS over diagnosis, exposing patients to unnecessary, expensive and sometimes dangerous therapy.* |
| 08:30-08:40 | Host: |
| 08:40-08:55 | Yes: |
| 08:55-09:10 | No: **Christopher Hawkes**, UK |
| 09:10-09:20 | Discussion and rebuttals |
| **09:20-10:10** | **Does OCT make VEP redundant?**<br>*Capsule: Visual evoked potentials (VEP) have traditionally been used to support the existence of subclinical involvement of the optic nerve in MS patients. The newly developed optical coherence tomography (OCT) is sensitive to anatomical changes in the retina and optic nerve. Does the OCT make the VEP obsolete or does the physiological measure add important information?* |
| 09:20-09:30 | Host: **Abhijit Chaudhuri**, UK |
| 09:30-09:45 | Yes: |
| 09:45-10:00 | No: |
| 10:00-10:10 | Discussion and rebuttals |
| **10:10-10:25** | *Coffee Break* |
| **08:30-10:10** | **THERAPY** |
| **10:25-11:15** | **Should new therapies for MS be used even with poor scientific support?**<br>*Capsule: Over the past three decades, numerous drugs were approved for MS, Following rigorous and expensive studies. In this session, the debaters will outline the pros and cons of using interventions based on poor scientific evidence, such as high dose vitamin D or hyperbaric oxygen (HBO).* |
| 10:25-10:35 | Host: **Olaf Stuve**, USA |
| 10:35-10:50 | Yes: |
| 10:50-11:05 | No: |
| 11:05-11:15 | Discussion and rebuttals |
| **11:15-12:05** | **Newly diagnosed MS patients should be started on aggressive therapy.**<br>*Capsule: Early treatment is claimed to improve long-term prognosis in MS. Recent studies also suggest that early aggressive therapy with potent immunosuppressive drugs ("induction therapy") may improve long-term outcomes and perhaps lower the risk of conversion to secondary-progressive MS. Should newly diagnosed MS patients be started on such aggressive therapies? Do the potential benefits always outweigh their risks?* |
| 11:15-11:25 | Host: |
| 11:25-11:40 | Yes: |
| 11:40-11:55 | No: **Uros Rot**, Slovenia |
| 11:55-12:05 | Discussion and rebuttals |

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| **12:05-12:15** | ***Short Break*** |
| **12:15-13:15** | **Industry Supported Symposium** |
| **13:15-14:15** | ***Lunch Break*** |
| **14:15-15:45** | **DISEASE COURSE** |
| **14:15-15:00** | **Are MS patients at increased risk for developing cancer?**<br>*Capsule: Whether people with MS are at higher risk of developing cancer has not been definitively established. The increased rate of general comorbidity would indicate a higher risk of cancer as well. However, some registers have not found an association. Could it be that there is higher risk of specific cancers, but not all cancers? And can newer highly potent immunosuppressive treatments modify the long term risk?* |
| 14:15-14:25 | Host: **Cris Constantinescu**, UK |
| 14:25-14:40 | Yes: |
| 14:40-14:55 | No: |
| 14:55-15:00 | Discussion and rebuttals |
| **15:00-15:45** | **MS is a primary progressive disease in all cases, but some patients have superimposed relapses.**<br>*Capsule: Patients with clinically isolated syndrome have been shown to have significant cortical changes in their brains. Subcortical asymptomatic alterations have also been described. Does that mean that MS is basically a degenerative disease with superimposed clinical flare-ups ("relapses") as epiphenomena or is MS an inflammatory disease of the brain with only secondary degeneration?* |
| 15:00-15:10 | Host: **Bart van Wijmeersch**, Belgium |
| 15:10-15:25 | Yes: **Antonio Scalfari**, UK |
| 15:25-15:40 | No: |
| 15:40-15:45 | Discussion and rebuttals |
| **15:45-16:00** | ***Coffee Break*** |
| **16:00-18:45** | **COGNITION IN MS AND FAKE NEWS** |
| **16:00-16:55** | **We are well enough equipped to identify fake news in MS therapy before they can cause harm.**<br>*Capsule: Fake news are news stories or hoaxes created to deliberately misinform or deceive readers. Information that patients with MS read online, and especially in their social media feeds is often inaccurate or untrue. Misinformation about MS therapies have also been disseminated to care providers.* |
| 16:00-16:10 | Host: |
| 16:10-16:25 | Yes: |
| 16:25-16:40 | No: |
| 16:40-16:55 | Discussion and rebuttals |
| **16:55-17:50** | **Cognitive decline is sufficient to define transition to secondary progressive multiple sclerosis (SPMS).**<br>*Capsule: There is no biomarker that indicates when a patient has transitioned from relapsing-remitting MS (RRMS) to SPMS, and consequently SPMS is a retrospective diagnosis, based primarily on motor disability. The period of diagnostic uncertainty separating RRMS and SPMS diagnoses often lasts years. Is cognitive decline sufficient to define this change?* |
| 16:55-17:05 | Host: **Antonio Scalfari**, UK |
| 17:05-17:20 | Yes: |
| 17:20-17:35 | No: |
| 17:35-17:50 | Discussion and rebuttals |
| **17:50-18:45** | **In MS patients with significant cognitive decline, drug treatment should be enhanced.**<br>*Capsule: Approximately 50% of people with MS become unemployed with a median EDSS of 3.0-3.5. They usually acquired hidden disabilities related to cognitive impairment. Should MS specific drug treatment be modified in patients with cognitive decline whose EDSS is otherwise unchanged?* |
| 17:50-18:00 | Host: **Laszlo Vecsei**, Hungary |
| 18:00-18:15 | Switch to a newer agent: |
| 18:15-18:30 | Not so fast: **Amos Korczyn**, Israel |

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| 18:30-18:45 | Discussion and rebuttals |

**HEADACHE**

| **Saturday October 3, 2020** | |
|---|---|
| **07:30-08:30** | **E-Poster Presentations (Exhibition Area)** |
| **08:30-10:10** | **SLEEP AND HORMONES** |
| **08:30-09:20** | **Estrogen containing contraceptives are safe in women with migraine with aura.**<br>*Capsule: Migraine with aura has been associated with increased risk of ischemic stroke in women. Prior studies have shown a further increase in risk in women using combined hormonal contraceptives (CHCs). This has led to guidelines recommending against use of CHCs in this population. Should these recommendations be changed?* |
| 08:30-08:40 | Host: **Pooja Dassan**, UK |
| 08:40-08:55 | Yes: |
| 08:55-09:10 | No: **Christopher Gottschalk**, USA |
| 09:10-09:20 | Discussion and rebuttals |
| **09:20-10:10** | **Correcting the derangement in sleep architecture is sufficient to treat cluster and migraine headache without medication.**<br>*Capsule: Migraines and cluster headache patients who do not sleep well develop more frequent and severe headaches. Would optimal sleep therapies ever be good enough to take the place of medication for the treatment of these headaches, or is sleep impairment just an epiphenomenon?* |
| 09:20-09:30 | Host: |
| 09:30-09:45 | Yes: **Bojana Zvan**, Slovenia |
| 09:45-10:00 | No: |
| 10:00-10:10 | Discussion and rebuttals |
| **10:10-10:25** | ***Coffee Break*** |
| **10:25-12:05** | **CGRP mAb's AND MIGRAINE PREVENTION** |
| **10:25-11:10** | **Peripheral trigeminal structures are the primary interaction site for CGRP antagonism in migraine prevention.**<br>*Capsule: CGRP is known to be widely distributed in the central and peripheral nervous system. The exact site of action of the mAbs to CGRP or its receptor is unknown, although several studies have been done.* |
| 10:25-10:35 | Host: **Fayyaz Ahmed**, UK |
| 10:35-10:50 | Yes: **Lars Edvinsson**, Sweden |
| 10:50-11:05 | No: **Dimos Mitsikostas**, Greece |
| 11:05-11:10 | Discussion and rebuttals |
| **11:10-12:05** | **The safety and efficacy of CGRP mAbs are known well enough for physicians to recommend them for long-term use.**<br>*Capsule: CGRP is a potent vasodilator and there was early concern about blocking it in patients that may have an impending stroke or myocardial infarction. CGRP is also involved in many other processes such as bone and wound healing as well as cardiovascular homeostasis and gastrointestinal function. Are these drugs safe enough?* |
| 11:10-11:20 | Host: |
| 11:20-11:35 | Yes: **Lars Edvinsson**, Sweden |
| 11:35-11:50 | No: **Fayyaz Ahmed**, UK |
| 11:50-11:55 | Discussion and rebuttals |
| **11:55-12:15** | **How are the CGRP monoclonal antibodies being used today?**<br>**Christopher Gottschalk**, USA |
| **12:15-13:15** | **Industry Supported Symposium** |
| **13:15-14:15** | ***Lunch Break*** |
| **13:15-14:15** | **Meet the Expert \|** |

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| **14:15-15:45** | **ATYPICAL MIGRAINE** |
| **14:15-15:00** | **Head injury can precipitate the onset of migraine**.<br>*Capsule: Post-trauma headache may occur in several phenotypes. Can a patient develop real migraine with or without aura, due to head trauma? This question raises clinical and legal issues.* |
| 14:15-14:25 | Host: |
| 14:25-14:40 | Yes: |
| 14:40-14:55 | No: |
| 14:55-15:00 | Discussion and rebuttals |
| **15:00-15:45** | **Vestibular migraine – does it exist?**<br>*Capsule: Vestibular migraine is a term used to describe episodic vertigo occurring in migraine patients; but should it be a distinct diagnosis, or simply a sensory manifestation, or even an aura, of migraine?* |
| 15:00-15:10 | Host: **Teena Shetty**, USA |
| 15:10-15:25 | Yes: **Christian Lampl**, Austria |
| 15:25-15:40 | No: |
| 15:40-15:45 | Discussion and rebuttals |
| **15:45-16:00** | ***Coffee Break*** |
| **16:00-19:00** | **NON-PHARMACOLOGICAL TREATMENTS IN HEADACHE** |
| **16:00-16:50** | **Headache devices will replace medications for the acute and preventive treatment of migraine and cluster headache.**<br>*Capsule: Headache devices are proliferating rapidly in the headache medicine field; there is hope that they will provide an alternative therapeutic option for patients with migraine and cluster headache. How strong is the evidence?* |
| 16:00-16:10 | Host: |
| 16:10-16:25 | Yes: |
| 16:25-16:40 | No: |
| 16:40-16:50 | Discussion and rebuttals |
| **16:50-17:40** | **Mushrooms extracts are a good treatment for chronic cluster headache.**<br>*Capsule: Psilocybin, lysergic acid diethylamide (LSD), and related psychedelic amines are reportedly effective for both preventive and acute treatment of cluster headache; but is there adequate scientific evidence to recommend it for our patients?* |
| 16:50-17:00 | Host: **Dimos Mitsikostas**, Greece |
| 17:00-17:15 | Yes: **Brian E. McGeeney**, USA |
| 17:15-17:30 | No: |
| 17:30-17:40 | Discussion and rebuttals |
| **17:40-18:20** | **The changing face of medication-overuse headache** |

**PARKINSON'S DISEASE (PD) I**

| **Saturday October 3, 2020** | |
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| **07:30-08:30** | **E-Poster Presentations (Exhibition Area)** |
| **08:30-10:10** | **TREATMENT OF OFF PERIODS** |
| **08:30-09:20** | **OFF episodes should be treated with on demand drugs before adjusting baseline treatments.**<br>*Capsule: OFF periods are significant problems in many PD patients. These can be treated by increasing the baseline dose of levodopa (which may cause peak dose dyskiaesias), or by on-demand non oral apomorphine, levodopa and like drugs. Which should be encouraged?* |
| 08:30-08:40 | Host: **Stuart Isaacson**, USA |
| 08:40-08:55 | Yes: |

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| 08:55-09:10 | Adjust baseline first: **Mark Lew**, USA |
| 09:10-09:20 | Discussion and rebuttals |
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| **09:20-10:10** | **Nondopaminergic therapies should be used before increasing levodopa to reduce OFF periods**<br>*Capsule: Despite extended-release formulations and adjunctive medications (MAO-B/COMT inhibitors, dopamine agonists), many patients continue to experience OFF time. Should non dopaminergic therapies be used before increasing levodopa?* |
| 09:20-09:30 | Host: |
| 09:30-09:45 | Yes: |
| 09:45-10:00 | No: |
| 10:00-10:10 | Discussion and rebuttals |
| **10:10-10:25** | ***Coffee Break*** |
| **10:25-12:05** | **NON MOTOR SYMPTOM TREATMENT** |
| **10:25-11:15** | **Should PD psychosis be treated if insight is retained?**<br>*Capsule: Psychosis is a significant problem in many PD patients at the advanced stages, particularly those suffering from cognitive decline. Treatment of psychosis may enhance their parkinsonism. Should it be given even if the patient maintains good insight?* |
| 10:25-10:35 | Host: |
| 10:35-10:50 | Yes: |
| 10:50-11:05 | No: **John Duda**, USA |
| 11:05-11:15 | Discussion and rebuttals |
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| **11:15-12:05** | **Botulinum toxin is first line treatment for sialorrhea in PD**<br>*Capsule:* Sialorrhea *is an inconvenient and embarrassing symptom which requires treatment. Is botulinum toxin the best approach?* |
| 11:15-11:25 | Host: **Mark Lew**, USA |
| 11:25-11:40 | Yes: |
| 11:40-11:55 | No: |
| 11:55-12:05 | Discussion and rebuttals |
| **12:05-12:15** | ***Short Break*** |
| **12:15-13:15** | **Industry Supported Symposium** |
| **13:15-14:15** | ***Lunch Break*** |
| **14:15-15:45** | |
| **14:15-14:55** | **Treatment goal should always center on continuous dopaminergic stimulation (CDS)**<br>*Capsule: Pulsatile stimulation of postsynaptic dopamine receptors has been associated with the pathogenesis and clinical emergence of motor complications. Should CDS be an early strategy?* |
| 14:15-14:25 | Host: **Mark Lew**, USA |
| 14:25-14:35 | Yes: **Stuart Isaacson**, USA |
| 14:35-14:45 | No: |
| 14:45-14:55 | Discussion and rebuttals |
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| **14:55-15:45** | **PET Imaging is an expensive tool with poor external validity and not relevant for future biomarker projects.**<br>*Capsule:* |
| 14:55-15:05 | Host: **John Duda**, USA |
| 15:05-15:20 | Yes: |
| 15:20-15:35 | No: |
| 15:35-15:45 | Discussion and rebuttals |
| **15:45-16:00** | ***Coffee Break*** |
| **16:00-19:00** | **NEUROSURGERY FOR MOVEMENT DISORDERS** |
| **16:00-17:00** | **Is there a role for neurosurgery in refractory Gilles de la Tourette syndrome (GdlT)?**<br>*Capsule: Neurosurgical advances in the treatment of movement disorders and psychiatric conditions continue to* |

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| | *grow and change the therapeutic scope. However, many questions remain about the safety, indications and long-term outcomes of neurosurgical interventions in GdIT patients who have recalcitrant motor and psychiatric symptoms undergoing neurosurgical procedures. Is there a role for neurosurgery in refractory GdIT?* |
| 16:00-16:10 | Host: **Nestor Galvez**, USA |
| 16:10-16:25 | Yes: **Patricia Limousin**, UK |
| 16:25-16:40 | No: |
| 16:40-17:00 | Discussion and rebuttals |
| **17:00-18:00** | **Is there a role for stereotactic ablation in movement disorders in the DBS era?**<br>*Capsule: DBS was introduced as an alternative to ablative therapy for tremor. The reversibility of DBS and the ability to adjust the implanted stimulator was appealing. But over time the invasiveness, adverse events profile and the high cost of the procedure became apparent. The introduction of minimally invasive ablative treatments such as MRI guided focused ultrasound has raised the question of whether it is time to reintroduce ablative procedures as an alternative to DBS.* |
| 17:00-17:10 | Host: **Ilana Schlesinger**, Israel |
| 17:10-17:25 | Yes: **John Duda**, USA |
| 17:25-17:40 | No: **Abdelhamid Benazzouz**, France |
| 17:40-18:00 | Discussion and rebuttals |

**ALZHEIMER'S DISEASE (AD) AND DEMENTIA**

| **Saturday October 3, 2020** | |
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| **07:30-08:30** | **E-Poster Presentations (Exhibition Area)** |
| **08:30-10:10** | **PRECLINICAL AND EARLY AD** |
| **08:30-09:20** | **Is subjective cognitive impairment itself a prelude to dementia?**<br>*Capsule: In a chronic medical condition, early diagnosis becomes an issue when treatment is available that can alter its course. Regarding AD, there is hope that novel prevention strategies will have the capacity of slowing down the neurodegeneration. Such treatments may provide greatest benefit to patients at the stage of absent or minor cognitive impairment. This debate will focus on the central question, can (and should) AD be diagnosed in the stage of subjective cognitive deficits, although disease-modifying interventions are still unproven?* |
| 08:30-08:40 | Host: |
| 08:40-08:55 | Yes: |
| 08:55-09:10 | No: **Panteleimon Giannakopoulos**, Switzerland |
| 09:10-09:20 | Discussion and rebuttals |
| **09:20-10:10** | **Is cognitive reserve just a buzzword lacking scientific value?**<br>*Capsule: The concept of reserve was established to account for the observation that a given degree of neurodegenerative pathology may result in varying degrees of symptoms in different individuals. There is a large amount of evidence on risk and protective factors for neurodegenerative diseases and dementia, yet the biological mechanisms that underpin the protective effects of certain lifestyle and physiological variables remain poorly understood, limiting the development of more effective strategies. This debate will focus on the important question, is reserve just another buzzword or is the phenomenon supported by convincing scientific evidence.* |
| 09:20-09:30 | Host: **Robert Perneczky**, Germany |
| 09:30-09:45 | Yes: **Panteleimon Giannakopoulos**, Switzerland |
| 09:45-10:00 | No: **Irena Rektorova**, Czech Republic |
| 10:00-10:10 | Discussion and rebuttals |
| **10:10-10:25** | ***Coffee Break*** |
| **10:25-12:05** | **PATHOPHYSIOLOGY OF AD** |
| **10:25-11:15** | **The development in understanding AD has not made an impact on patient care.**<br>*Capsule: AD is characterized by cognitive deterioration, but non-cognitive behavioral symptoms are also frequent and often associated with more suffering than cognitive decline. Treatment of those symptoms may be difficult and challenging, and there is insufficient evidence to support treatment decisions. This debate will focus on the question whether the improved understanding of AD pathology has improved patient care and treatment, including non-cognitive symptoms.* |

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| 10:25-10:35 | Host: **Ruth Itzhaki**, UK |
| 10:35-10:50 | Yes: **Judith Aharon**, Israel |
| 10:50-11:05 | No: |
| 11:05-11:15 | Discussion and rebuttals |
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| **11:15-12:05** | **Vascular lesions contribute directly to AD pathology.**<br>*Capsule: Over several years, evidence has been accumulating that suggests vascular disease impacts on common causes of dementia, in particular AD. Modifiable vascular risk factors such as hypertension, diabetes, dyslipidemia and adiposity are all linked to AD. However, it is unclear whether these factors contribute to or promote AD pathology itself or just add vascular damage.* |
| 11:15-11:25 | Host: |
| 11:25-11:40 | Yes: **Raj Kalaria**, UK |
| 11:40-11:55 | No: |
| 11:55-12:05 | Discussion and rebuttals |
| **12:05-12:15** | ***Short Break*** |
| **12:15-13:15** | **Industry Supported Symposium** |
| **13:15-14:15** | ***Lunch Break*** |
| **13:15-14:15** | **Meet the Expert \|** |
| **14:15-15:45** | **RISK AND PROTECTIVE FACTORS** |
| **14:15-15:00** | **Does aerobic exercise protect cognition?**<br>*Capsule: Lifestyle changes have been suggested for dementia prevention. Physical activity engagement has repeatedly been associated with preserved cognition and lower risk for cognitive decline and dementia among older adults. Whether physical activity is neuroprotective by itself or if it mitigates enhanced risk for dementia via other factors is less well understood. This debate will discuss whether physical activity protects cognitive function and whether we know enough about the phenomenon to design effective interventions.* |
| 14:15-14:25 | Host: |
| 14:25-14:40 | Yes: |
| 14:40-14:55 | No: **Naji Tabet**, UK |
| 14:55-15:00 | Discussion and rebuttals |
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| **15:00-15:45** | **Is deafness a causative risk factor to dementia?**<br>*Capsule: Hearing loss is associated with increased risk for dementia. Other data suggest that there may be a causal link between deafness and cognitive decline or that hearing loss may merely be an early symptom of neurodegenerative changes.* |
| 15:00-15:10 | Host: **Gill Livingston**, UK |
| 15:10-15:25 | Yes: **Sergi Costafreda**, UK |
| 15:25-15:40 | No: |
| 15:40-15:45 | Discussion and rebuttals |
| **15:45-16:00** | ***Coffee Break*** |
| **16:00-17:30** | **NEW DEFINITIONS AND APPROACHES** |
| **16:00-16:45** | **Is the new NIA-AA research definition of AD helpful?**<br>*Capsule: The 2011 NIA-AA definition of AD was based on clinical symptoms. However, the new 2018 definition eliminates the use of clinical phenotypes and rather depends on biological manifestations like whether amyloid-β (Aβ) and tau pathology or neuroimaging evidence of neurodegeneration exist, regardless of clinical manifestations. Is this change useful?* |
| 16:00-16:10 | Host: **Edson Amaro**, Brazil |
| 16:10-16:25 | Yes: |
| 16:25-16:40 | No: **Mee Park**, South Korea |
| 16:40-16:45 | Discussion and rebuttals |

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| **16:45-17:30** | **Will big data help us to find cure for dementia?**<br>*Capsule: In recent years there has been a surge towards big data approaches in AD, following a general trend in other disciplines. However, not everyone is convinced and the debate on the merits of big data for identifying effective treatments for AD is in full swing. That big collections might generally be useful is not the issue. It was suggested that we can let the data speak for themselves. But what does the amassed data actually explain about the underlying pathophysiology and how can it help us identify new drug targets? This debate will focus on the promise of big data initiatives for finding a cure for AD.* |
| 16:45-16:55 | Host: **Stefano Sensi**, Italy |
| 16:55-17:10 | Yes: |
| 17:10-17:25 | No: **Peter Whitehouse**, USA |
| 17:25-17:30 | Discussion and rebuttals |
| **17:30-19:00** | **FRONTOTEMPORAL DEMENTIA (FTD)** |
| **17:30-18:15** | **The term Frontotemporal Dementia (FTD) is no longer of value.**<br>*Capsule: FTD is the second most common cause of young-onset dementia, which includes several distinct, heterogeneous sub-syndromes. FTD is characterized by progressive deficits in behavior, language, and executive function, but individual symptoms vary considerably and the underlying pathology is also heterogeneous. Over the last decades, the nomenclature of this group of disorders has undergone several iterations. This debate focusses on the question whether the term FTD is still relevant or if it should be replaced.* |
| 17:30-17:40 | Host: |
| 17:40-17:55 | Yes: |
| 17:55-18:10 | No: **James Rowe**, UK |
| 18:10-18:15 | Discussion and rebuttals |
| **18:15-19:00** | **All patients with a diagnosis of FTD should have genetic testing.**<br>*Capsule: Approximately 40 percent of affected individuals with FTD have a family history that includes at least one other relative diagnosed with a neurodegenerative disorder. Three genes account for the majority of mutation-associated hereditary FTD cases, including C9orf72, GRN and MAPT and others. Furthermore, a very small percentage of people with sporadic FTD have a mutation in a known FTD gene. This debate focuses on the question if this knowledge about the genetics of FTD is reason enough to perform genetic testing (and counselling) in all patients diagnosed with the disease.* |
| 18:15-18:25 | Host: **Robert Perneczky**, Germany |
| 18:25-18:40 | Yes: **Lea Grinberg**, Brazil/USA |
| 18:40-18:55 | No: |
| 18:55-19:00 | Discussion and rebuttals |

**STROKE**

| **Saturday October 3, 2020** | |
|---|---|
| **07:30-08:30** | **E-Poster Presentations (Exhibition Area)** |
| **08:30-10:10** | **NEUROIMAGING IN ACUTE ISCHEMIC STROKE** |
| **08:30-09:20** | **Is penumbral imaging mandatory for potential thrombectomy in patients arriving beyond six hours?**<br>*Capsule: There is general agreement amongst stroke experts that patient selection is essential for successful thrombectomy. The introduction of penumbral imaging may allow for improved patient evaluation but comes at a higher cost. Is there sufficient evidence that such imaging is made mandatory prior to initiation of treatment?* |
| 08:30-08:40 | Host: **Joanna Wojczal**, Poland |
| 08:40-08:55 | Yes: |
| 08:55-09:10 | No: **Ashfaq Shuaib**, Canada |
| 09:10-09:20 | Discussion and rebuttals |
| **09:20-10:10** | **Do diffusion weighted imaging (DWI) negative strokes exist?**<br>*Capsule: Stroke is a clinical entity. Its exact identification is crucial as therapeutic options nowadays are associated with some risks. DWI MRI is considered the best imaging technique for the confirmation of acute ischemic stroke (AIS). Sensitivity, however, is not perfect, with debatable underlying reasons, raising the question: Do AIS with* |

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| | *negative DWI imaging really exist?* |
| 09:20-09:30 | Host: **Olena Tsurkalenko**, Ukraine |
| 09:30-09:45 | Yes: |
| 09:45-10:00 | No: |
| 10:00-10:10 | Discussion and rebuttals |
| **10:10-10:25** | ***Coffee Break*** |
| **10:25-12:05** | **HEART AND BRAIN** |
| **10:25-11:15** | **Should all patients with embolic stroke of undetermined source (ESUS) be anticoagulated?** |
| | *Capsule: ESUS patients are more likely to have a cardioembolic source of stroke, so may benefit from anticoagulation. However, studies to date have not supported anticoagulation in all patients with ESUS. Are there robust reasons that they should be anticoagulated?* |
| 10:25-10:35 | Host: |
| 10:35-10:50 | Yes: |
| 10:50-11:05 | No: |
| 11:05-11:15 | Discussion and rebuttals |
| **11:15-12:05** | **Is left atrial appendage closure underutilized for stroke prevention in atrial fibrillation?** |
| | *Capsule: The majority of embolic strokes patients with nonvalvular atrial fibrillation are associated with left atrial thrombi, and left atrial appendage closure may be a suitable alterative to chronic anticoagulation.* |
| 11:15-11:25 | Host: |
| 11:25-11:40 | Yes: |
| 11:40-11:55 | No: **Roni Eichel**, Israel |
| 11:55-12:05 | Discussion and rebuttals |
| **12:05-12:15** | ***Short Break*** |
| **12:15-13:15** | **Industry Supported Symposium** |
| **13:15-14:15** | ***Lunch Break*** |
| **13:15-14:15** | **Meet the Expert \|** |
| **14:15-15:45** | **ACUTE ISCHEMIC STROKE (AIS) MANAGEMENT** |
| **14:15-15:00** | **Mobile stroke units (MSU) are useful and cost effective for patients with AIS.** |
| | *Capsule: IV tPA was approved as an effective treatment for AIS within 4.5 hours. It also was shown that the sooner the tPA is administered the better are the chances of beneficial outcome – "Time is Brain". Therefore, MSU with CT scan were introduced with the ability to give tPA in the ambulance and by that to save time. It is still unsettled whether MSU actually have an impact on patients' outcome and are cost effective. This debate will try to shed light on this controversial issue.* |
| 14:15-14:25 | Host: **Joanna Wojczal**, Poland |
| 14:25-14:40 | Yes: **Silke Walter**, Germany |
| 14:40-14:55 | No: |
| 14:55-15:00 | Discussion and rebuttals |
| **15:00-15:45** | **Does the main benefit of AIS treatment come from tPA or stroke unit care?** |
| | *Capsule: The presence of a dedicated stroke unit allows for the management of all patients with suspected AIS. Treatment with tPA can only be offered to a smaller subset of AIS patients but the improvement in some treated patients can be very significant. In an era of limited resources, should we focus on ensuring that all AIS patients be admitted to a stroke unit or recommend fast triage methods for timely thrombolysis?* |
| 15:00-15:10 | Host: |
| 15:10-15:25 | tPA: |
| 15:25-15:40 | Stroke unit: **Ashfaq Shuaib**, Canada |
| 15:40-15:45 | Discussion and rebuttals |
| **15:45-16:00** | ***Coffee Break*** |
| **16:00-17:30** | **SECONDARY STROKE PREVENTION** |
| **16:00-16:45** | **Should statins be given to people over age 80 for stroke prevention?** |
| | *Capsule: There is considerable evidence that the use of statins results in reduction of cardiovascular morbidity and* |

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| | *mortality. Long-term treatment with statins can lead to side effects including muscle and liver damage. Clinical trials evaluating the efficacy of statins have mostly enrolled subjects less than 75 years of age. Can we extrapolate the evidence to older individuals in whom the risk of side-effects may be higher?* |
| 16:00-16:10 | Host: |
| 16:10-16:25 | Yes: |
| 16:25-16:40 | No: **Vida Demarin**, Croatia |
| 16:40-16:45 | Discussion and rebuttals |
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| **16:45-17:30** | **Should symptomatic extracranial vertebral artery stenosis be stented?**<br>*Capsule: Stenosis in the vertebro-basilar system accounts for about one quarter of all posterior circulation strokes. The risk profile is similar to that seen for carotid stenosis. Recent phase 2 trials have shown that extracranial vertebral stenosis can be stented with low risk but whether this reduces recurrent stroke risk compared with best medical therapy alone remains controversial. The debate will consider whether based on current evidence stenting should be recommended for recently symptomatic extracranial vertebral artery stenosis.* |
| 16:45-16:55 | Host: **Hugh Markus**, UK |
| 16:55-17:10 | Yes: **Laszlo Csiba**, Hungary |
| 17:10-17:25 | No: **Hrvoje Budincevic**, Croatia |
| 17:25-17:30 | Discussion and rebuttals |
| **17:30-19:00** | **ANTITHROMBOTIC TREATMENTS** |
| **17:30-18:15** | **Should TIA patients be routinely treated chronically with both aspirin and clopidogrel?**<br>*Capsule: In some studies, dual antiplatelet therapy has benefits in the short term compared to single agents. However, the duration of benefit may be limited, and there may be some patients who would not benefit. In addition, dual therapy carries risk of complications, particularly hemorrhage. Is there sufficient evidence to recommend long-term dual antiplatelet therapy for all patients with TIA or minor strokes?* |
| 17:30-17:40 | Host: |
| 17:40-17:55 | Yes: |
| 17:55-18:10 | No: |
| 18:10-18:15 | Discussion and rebuttals |
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| **18:15-19:00** | **In the presence of cerebral microbleeds (CMBs), antithombotic therapy should be avoided.**<br>*Capsule: The presence of microbleeds (detected only with MRI) is associated with increased risk of hemorrhagic and perhaps of ischemic stroke. The risk depends on the location and number of microbleeds. How dangerous is antithrombotic therapy in patients with microbleeds? The session provides an overview about the pros and cons.* |
| 18:15-18:25 | Host: |
| 18:25-18:40 | Yes: |
| 18:40-18:55 | No: **Laszlo Csiba**, Hungary |
| 18:55-19:00 | Discussion and rebuttals |

**PARKINSON'S DISEASE (PD) II**

| **Sunday October 4, 2020** | |
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| **07:00-08:00** | **E-Poster Presentations (Exhibition Area)** |
| **08:00-08:30** | **EXCELLENCE IN NEUROLOGY AWARD** |
| **08:00-08:30** | **Presentation of the CONy "Excellence in Neurology" Award**<br>Awardee presented by<br>**Cris Constantinescu**<br><br>Award given by<br>**Abhijit Chaudhuri**<br><br>Awardee |
| **08:30-10:10** | **DEEP BRAIN STIMULATION (DBS)** |
| **08:30-09:20** | **When is the right time to refer PD patients for deep brain stimulation (DBS)?** |

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| | *Capsule: DBS is effective in treating medication-refractory symptoms (motor and non-motor) and improves patients' quality of life in advanced PD. Currently, it is usually performed in late stage of PD. Advances in our understanding of the natural history of the disease, improved surgical techniques, brain imaging and device design require us to re-evaluate whether the right time for DBS is in earlier stages of the disease.* |
| 08:30-08:40 | Host: **Mike Samuel**, UK |
| 08:40-08:55 | Late: **Patricia Limousin**, UK |
| 08:55-09:10 | Early: **Vladimira Vuletic**, Croatia |
| 09:10-09:20 | Discussion and rebuttals |
| **09:20-10:10** | **DBS effectiveness against nonmotor features in PD is similar to those of infusion therapies.** |
| | *Capsule: DBS and infusion therapies are both recognized as therapeutic approaches in the treatment of motor symptoms in advanced stage PD when patients develop "wearing off" and/or dyskinesias with oral dopaminergic medication. However, the effects of these two therapeutic approaches on nonmotor features are still under debate.* |
| 09:20-09:30 | Host: |
| 09:30-09:45 | Yes: |
| 09:45-10:00 | No: **Keyoumars Ashkan**, UK |
| 10:00-10:10 | Discussion and rebuttals |
| **10:10-10:25** | ***Coffee Break*** |
| **10:25-12:05** | **CONTROVERSIES IN CLINICAL APPROACH** |
| **10:25-11:15** | **Clinical assessment in PD - Motor assessment is the key and nonmotor is marginal.** |
| | *Capsule: PD is primarily a motor disorder, yet non-motor symptoms (NMS) become more widely recognized. How important are these NMS in the clinical assessment of the patient?* |
| 10:25-10:35 | Host: **Gennaro Pagano**, UK |
| 10:35-10:50 | Yes: **Edoardo de Natale**, UK |
| 10:50-11:05 | No: |
| 11:05-11:15 | Discussion and rebuttals |
| **11:15-12:05** | **Should non troublesome dyskinesias be treated?** |
| | *Capsule: Dyskinesias occur frequently in PD patients treated with dopaminergic medications. The dyskinesia frequently is troubling to spouses or caregivers more than to the patients themselves, who may be unaware of their impact in daily activities. Should these patients still be treated?* |
| 11:15-11:25 | Host: |
| 11:25-11:40 | Yes: |
| 11:40-11:55 | No: |
| 11:55-12:05 | Discussion and rebuttals |
| **12:05-12:15** | ***Short Break*** |
| **13:15-14:15** | ***Lunch Break*** |
| **14:15-15:45** | **GENES AND ENVIRONMENT** |
| **14:15-15:00** | **Should PD patients carrying GBA mutations be treated differently from gene mutation negatives?** |
| | *Capsule: GBA mutations acting through lysosomal pathways are known to contribute to a minority PD cases. Should there be an attempt to activate lysosomes in order to protect against PD?* |
| 14:15-14:25 | Host: **Vladimira Vuletic**, Croatia |
| 14:25-14:40 | Yes: **Anthony Schapira**, UK |
| 14:40-14:55 | No: |
| 14:55-15:00 | Discussion and rebuttals |
| **15:00-15:45** | **Are there important environmental factors for PD?** |
| | *Capsule: The pathogenesis of the neurodegenerative processes in PD are not well understood. Although several genes were found to be associated with the development of PD, the causative agents for a great percent of cases remain unclear. Various factors were incriminated to increase or reduce the risk of PD development yet their contribution as well as interactions with genetic factors are unknown.* |

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| 15:00-15:10 | Host: **Alastair Noyce**, UK |
| 15:10-15:25 | Yes: |
| 15:25-15:40 | No: **Zvezdan Pirtosek**, Slovenia |
| 15:40-15:45 | Discussion and rebuttals |
| **15:45-16:00** | ***Coffee Break*** |
| **16:00-19:00** | **CONTROVERSIES IN PD ETIOPATHOGENESIS** |
| **16:00-16:50** | **Is CSF alpha-snyuclein a useful biomarker for PD?**<br>*Capsule: A change in the content of α-synuclein (α-syn) in CSF is considered as a promising biomarker of PD. Indeed, the total α-syn content in CSF decreases, and the fractions of phosphorylated and oligomeric α-syn increase in PD. However, attempts to use α-syn as a biomarker for differential diagnosis or prognosis were unsuccessful so far. Can CSF α-syn be used as a biomarker?* |
| 16:00-16:10 | Host: **Michael Ugrumov**, Russia |
| 16:10-16:25 | Yes: |
| 16:25-16:40 | No: **Mark Lew**, USA |
| 16:40-16:50 | Discussion and rebuttals |
| **16:50-17:40** | **Should continuous dopaminergic stimulation replace pulsatile once motor fluctuations develop?**<br>*Capsule: Dopaminergic stimulation is the key treatment for PD. However, it is still debatable whether pulsatile stimulation contributes to motor complication. Should patients be treated with continuous therapies early when OFF and dyskinesia emerge?* |
| 16:50-17:00 | Host: |
| 17:00-17:15 | Yes: |
| 17:15-17:30 | No: |
| 17:30-17:40 | Discussion and rebuttals |
| 17:40-18:10 | The personalized Parkinson's |
| 18:10-18:15 | Discussion |
| **18:15-19:00** | **CLINICAL PATHOLOGICAL CONFERENCE (CPC)** |
| **18:15-19:00** | **Case Presentation - A young man with parkinsonism**<br>Host: **Tom Warner**, UK<br>Clinical: **Helen Ling**, UK<br>Pathology:<br>Discussion |

**EPILEPSY**

| **Sunday October 4, 2020** | |
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| **07:30-08:30** | **E-Poster Presentations (Exhibition Area)** |
| **08:30-10:10** | **GENERAL EPILEPSY** |
| **08:30-09:20** | **Ambulatory video-EEG monitoring can replace in-hospital video-EEG.**<br>*Capsule: Outpatient ambulatory video-EEG devices are now widely available. Are they a reasonable substitute for inpatient monitoring? Can they provide the same information? Should this be done first before considering in hospital assessment? What are the risks involved?* |
| 08:30-08:40 | Host: |
| 08:40-08:55 | Yes: **Antonio Gil-Nagel**, Spain |
| 08:55-09:10 | No: **Ilan Blatt**, Israel |
| 09:10-09:20 | Discussion and rebuttals |
| **09:20-10:10** | **Combination antiepileptic drug (AED) therapy should be offered immediately after failure of a single antiepileptic drug.** |

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| | *Capsule: Though monotherapy has advantages, there is clear evidence from randomized trials that polytherapy affords improvement to many patients, and reduces mortality as well. If combination therapy is used, when should it be implemented?* |
| 09:20-09:30 | Host: |
| 09:30-09:45 | Yes: **Martin Brodie**, UK |
| 09:45-10:00 | No: |
| 10:00-10:10 | Discussion and rebuttals |
| **10:10-10:25** | ***Coffee Break*** |
| **10:25-12:05** | **PSYCHIATRY IN EPILEPSY** |
| **10:25-11:15** | **Antidepressant drugs should be avoided if possible in epilepsy.** |
| | *Capsule: Many antidepressant medications can provoke seizures in animals, and concerns have been raised that these drugs may trigger seizures in some patients. Is the efficacy of these agents sufficient to warrant their use, given potential risks?* |
| 10:25-10:35 | Host: **Alla Guekht**, Russia |
| 10:35-10:50 | Yes: **Ilan Blatt**, Israel |
| 10:50-11:05 | No: **William Theodore**, USA |
| 11:05-11:15 | Discussion and rebuttals |
| **11:15-12:05** | **Psychotherapy improves outcome in "psychogenic" seizures.** |
| | *Capsule: In patients with "psychogenic" seizures, spontaneous remission rates are quite high and patient adherence to therapy quite low. Is there evidence that psychotherapy provides long-term benefit?* |
| 11:15-11:25 | Host: **Marco Mula**, UK |
| 11:25-11:40 | Yes: **William Curt LaFrance**, USA |
| 11:40-11:55 | No: |
| 11:55-12:05 | Discussion and rebuttals |
| **12:05-12:15** | ***Short Break*** |
| **12:15-13:15** | **Industry Supported Symposium** |
| **13:15-14:15** | ***Lunch Break*** |
| **13:15-14:15** | **Meet the Expert** |
| **14:15-15:45** | **STATUS EPILEPTICUS (SE)** |
| **14:15-15:00** | **Combination therapy should be used as first line treatment for status epilepticus (SE).** |
| | *Capsule: Success rates diminish for treating SE with failure of each successive drug that is administered. Furthermore, the longer seizures last, the harder it is to control them. Can we improve outcome by aggressively using polypharmacy as initial therapy in SE?* |
| 14:15-14:25 | Host: **John Duncan**, UK |
| 14:25-14:40 | Yes: **Matthew Walker**, UK |
| 14:40-14:55 | No: **Alla Guekht**, Russia |
| 14:55-15:00 | Discussion and rebuttals |
| **15:00-15:45** | **Cryptogenic SE should be treated with immunomodulation as soon as it is diagnosed.** |
| | *Capsule: NORSE and FIRES are epilepsy syndromes resistant to treatment with conventional AED and may require immune modulation for cessation of seizures. Should patients be presumptively treated with immunosuppressive agents early in the course of illness when status epilepticus has no known cause?* |
| 15:00-15:10 | Host: **Joanna Jedrzejczak**, Poland |
| 15:10-15:25 | Yes: |
| 15:25-15:40 | No: **Matthias Koepp**, UK |
| 15:40-15:45 | Discussion and rebuttals |
| **15:45-16:00** | ***Coffee Break*** |
| **16:00-19:00** | **EPILEPSY THERAPY** |

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| **16:00-17:20** | **Epilepsy Cases –**<br>Challenging diagnostic and management cases will be presented to the faculty and audience for discussion. A lively debate is anticipated for each case.<br>**Michael Sperling**, USA |
| **17:20-18:10** | **Should surgery be offered to patients after failure of two AED?**<br>*Capsule: Epidemiological studies suggest that drug failure is quite likely once two agents have failed to control seizures. On the other hand, the literature contains numerous reports of response to drug therapy in patients formerly considered drug resistant. Are the ILAE guidelines supported by the evidence?* |
| 17:20-17:30 | Host: **Manjari Tripathi**, India |
| 17:30-17:45 | Yes: **Zeljka Petelin Gadže**, Croatia |
| 17:45-18:00 | No: |
| 18:00-18:10 | Discussion and rebuttals |
| **18:10-19:00** | **The newer AED are more effective than the established ones.**<br>*Capsule: Over the past 20 years a number of new antiseizure drugs have been introduced around the world as adjunctive treatment and subsequently as monotherapy for pharmacoresistant and newly diagnosed epilepsy. Have they improved overall outcomes in terms of seizure freedom and so proved value for money?* |
| 18:10-18:20 | Host: |
| 18:20-18:35 | Yes: **Andreas Schulz-Bonhage**, Germany |
| 18:35-18:50 | No: **Martin Brodie**, UK |
| 18:50-19:00 | Discussion and rebuttals |

**DEMENTIA SATELLITE – OVERCOMING THE IMPASSE IN DEMENTIA PREVENTION AND TREATMENT**

| **Sunday October 4, 2020** | |
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| **07:30-08:30** | **E-Poster Presentations (Exhibition Area)** |
| **08:30-10:10** | **SECTION TITLE** |
| **08:30-08:45** | **Introduction from the Alzheimer's Association (AA)**<br>*Capsule: The AA leads the way to end Alzheimer's and all other dementia — by accelerating global research, driving risk reduction and early detection, and maximizing quality care and support. The AA fosters and facilitates advances in scientific discussion through publications, conferences and collaborations, including our partnership with CONy to support the CONy Dementia Satellite.* |
| **08:45-09:15** | **The complex reality**<br>*Capsule: The overwhelming failure of the attempts to prevent or cure Alzheimer's disease (AD) should lead us to rethink the problem. Is AD a real disease or is it a syndrome? Can we use genetic early onset AD as a template for the sporadic disease? What is the role of comorbidities in older people with dementia? Since AD is a multifactorial disorder can we expect to find a "silver bullet" that will cure AD? Is the idea of "the sooner the better" really valid?*<br>**Amos Korczyn**, Israel |
| 09:15-09:25 | Discussion |
| **09:25-09:55** | **Animal models of AD**<br>*Capsule: Is the employment of young, genetically modified rodents without comorbidities, likely to lead us to find a cure for sporadic AD? Several treatments were beneficial in those animal models yet failed in humans. Are the models misleading us? How can we improve?*<br>**Andrea Tenner**, USA |
| 09:55-10:10 | Discussion |
| **10:10-10:25** | ***Coffee Break*** |
| **10:25-13:15** | **INTERVENTIONS AND PATHOLOGY** |
| **10:25-10:55** | **Non-pharmacological interventions**<br>*Capsule: Are environmental interventions (optimal CV control, diet, physical activity, etc.) likely to change the frequency of dementia and by how much? Have any of these factors been shown to prevent the occurrence of dementia or do they merely delay the onset, and if so, by how much? What is the number needed to treat (NNT in* |

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| | *order to prevent one case? These are standard questions for drug treatment and should be also relevant for non-phrenological interventions?*<br>**Kristine Yaffe**, USA |
| 10:55-11:20 | Discussion |
| **11:20-11:50** | **Neuropathology of dementia**<br>*Capsule: What can pathology contribute to our understanding, given that autopsies come very late in the disease course and show mixed pathology in most cases?*<br>**Lea Grinberg**, Brazil/USA |
| 11:50-12:15 | Discussion |
| **12:15-13:15** | **Industry Symposium** |
| **13:15-14:15** | ***Lunch Break*** |
| **13:15-14:15** | **Meet the Expert** |
| **14:15-15:45** | **AMYLOID AND STUDIES** |
| **14:15-14:45** | **Amyloid in AD**<br>*Capsule: Are βA and tau wrong treatment targets in sporadic, late onset AD, given the disappointments with human studies, although they were successful in eliminating amyloid yet without clinical benefit? Since a deposition is very prevalent in aging and is not necessarily associated with dementia, how do we know that it is a worthwhile target? Could other biomarkers, such as synaptic loss or neurodegeneration add specificity?* |
| 14:45-15:00 | Discussion |
| **15:00-15:30** | **To futility or not – when and how should futility analysis be applied?**<br>*Capsule: AD treatment exploratory studies are excessively costly and long. Should the studies always be continued till the planned end? Is discontinuation always justified and ethical when treatment seems non-effective during the study?* |
| 15:30-15:45 | Discussion |
| **15:45-16:00** | ***Coffee Break*** |
| **16:00-19:00** | **AD THERAPY** |
| **16:00-16:30** | **Is APOE4 a potential treatment target, given that we do not understand its mechanism?**<br>*Capsule: It is now almost 30 years since the identification of APOE polymorphism as important genetic determinants of AD yet the underlying mechanism is still unknown and it is not even clear whether APOE4 is toxic or just less protective than APOE3. And should APOE4 related dementia be designated as a separate disease?*<br>**Daniel Michaelson**, Israel |
| 16:30-16:45 | Discussion |
| **16:45-17:15** | **Is neuroinflammation a useful potential therapeutic target?**<br>*Capsule: Examination by pathologists demonstrate the existence of inflammation in the brain of patients with dementia and this is supported by imaging, genetic, and neurochemical studies. However, attempts to ameliorate the condition of patients have largely failed. Does that mean that the inflammatory processes are just epiphenomena, or perhaps have different roles in early and late stages of the disease? Could it be that inflammation has both beneficial and toxic effects?*<br>**Michael Heneka**, Germany |
| 17:15-17:30 | Discussion |
| **17:30-18:00** | **Fear and loathing in AD trials**<br>*Capsule: The cases of Aducanumab, albumin/IVIG exchange, and oligomannurarate show the difficulties in preforming and interpreting data. What is the way forward? Are the targets wrong or are the other methods used mistaken? Failure of design, methods, execution or analysis?* |
| 18:00-18:15 | Discussion |
| **18:15-18:45** | **The need for multiple targets, outcomes, and approaches.**<br>*Capsule:* 16.2.20 |
| 18:45-19:00 | Discussion |

| **Sunday October 4, 2020** | |
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| **07:30-08:30** | **E-Poster Presentations (Exhibition Area)** |
| **08:30-10:10** | **QUESTIONABLE AUTOIMMUNE DISEASES** |
| **08:30-09:20** | **PANDAS is a clinically distinct entity and needs early and prompt immunotherapy.**<br>*Capsule: The nosology of pediatric acute neuropsychiatric syndromes like PANDAS remains controversial, and the evidence for immunopathology and in particular autoimmunity driven by streptococcal infection is still being questioned. Is PANDAS a distinct disorder and is prompt immunotherapy indicated for treatment?* |
| 08:30-08:40 | Host: |
| 08:40-08:55 | Yes: **Ronny Wickström**, Sweden |
| 08:55-09:10 | No: **Ming Lim**, UK |
| 09:10-09:20 | Discussion and rebuttals |
| **09:20-10:10** | **Is narcolepsy an autoimmune disorder which should be treated as such?**<br>*Capsule: Narcolepsy is a relatively rare disorder and its cause is unknown. There is a genetic component, particularly the HLA system, which is important in immune regulation. This leads to the hypothesis that interference with the immune system may be helpful in patents.* |
| 09:20-09:30 | Host: |
| 09:30-09:45 | Yes: **Jacek Losy**, Poland |
| 09:45-10:00 | No: |
| 10:00-10:10 | Discussion and rebuttals |
| **10:10-10:25** | ***Coffee Break*** |
| **10:25-12:05** | **NEUROMYELITIS OPTICA SPECTRUM DISORDER (NMOSD) TREATMENT** |
| **10:25-11:15** | **New regulatory-approved medications should be used exclusively for prevention of attacks of NMOSD over currently used non-regulatory approved medications.**<br>*Capsule: It is likely that 3 immunomodulatory treatments, a C5 complement inhibitor, an anti-CD19 monoclonal antibody and an inhibitor of IL6 receptor, will all receive regulatory approval for treatment of NMOSD. Do these drugs offer sufficient advantages that they should replace currently used immunotherapies that are widely regarded as effective and are less expensive?* |
| 10:25-10:35 | Host: |
| 10:35-10:50 | Yes: |
| 10:50-11:05 | No: |
| 11:05-11:15 | Discussion and rebuttals |
| **11:15-12:05** | **NMOSD attacks should be treated with apheresis/plasma exchange at first presentation, either with or without corticosteroids.**<br>*Capsule: Recent experience suggests that concomitant or first line treatment with plasma exchange may be superior to treatment with corticosteroids. Is a change in the standard approach of using corticosteroids first appropriate given the current state of knowledge?* |
| 11:15-11:25 | Host: **Bruno Gran**, UK |
| 11:25-11:40 | Yes: |
| 11:40-11:55 | Steroids should be used first: **Maria Isabel Leite**, UK |
| 11:55-12:05 | Discussion and rebuttals |
| **12:05-12:15** | ***Short Break*** |
| **12:15-13:15** | **Industry Symposium** |
| **13:15-14:15** | ***Lunch Break*** |
| **13:15-14:15** | **Meet the Expert** |
| **14:45-15:45** | **NMOSD AND CIDP** |
| **14:15-15:00** | **The 2015 International Panel criteria for NMOSD are outdated and should be replaced with a diagnostic classification based on autoantibody status rather than clinical presentation (*i.e.* AQP4 disease, MOG disease).**<br>*Capsule: We now know the molecular target of the autoimmune insult in the majority of patients with NMOSD, and molecular classification based on the target of the antibody can be used in lieu of clinical criteria for diagnosis of what we currently refer to as NMOSD. Are we ready for a molecular classification of NMOSD in 2020?* |
| 14:15-14:25 | Host: |

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| 14:25-14:40 | Yes: |
| 14:40-14:55 | No: |
| 14:55-15:00 | Discussion and rebuttals |
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| **15:00-15:45** | **Treatment of CIDP - immunoglobulins or steroids first?**<br>*Capsule: Many treatments have been advocated for CIDP, but the best accepted options are intravenous immunoglobulin and corticosteroids. Which treatment is best and should be applied first?* |
| 15:00-15:10 | Host: **Joab Chapman**, Israel |
| 15:10-15:25 | Immunoglobulins: |
| 15:25-15:40 | Steroids: **Eduardo Nobile-Orazio**, Italy |
| 15:40-15:45 | Discussion and rebuttals |
| **15:45-16:00** | ***Coffee Break*** |

**AMYOTROPHIC LATERAL SCLEROSIS (ALS)**

| **Sunday October 4, 2020** | |
|---|---|
| **07:30-08:30** | **E-Poster Presentations (Exhibition Area)** |
| **08:30-10:10** | **ALS AND ALS-PDC** |
| **08:30-09:20** | **ALS should not be considered a neuromuscular disorder.**<br>*Capsule: ALS is now recognized to have clinical, histopathological and genetic overlap with FTD. Brain-based pathology is consistently identifiable, yet ALS frequently continues to be classified alongside neuromuscular disorders of peripheral nerves, rather than cerebral neurodegenerative disorders, this may have a detrimental impact on research funding and restrict optimal collaboration.* |
| 08:30-08:40 | Host: **Giancarlo Logroscino**, Italy |
| 08:40-08:55 | Yes: |
| 08:55-09:10 | No: |
| 09:10-09:20 | Discussion and rebuttals |
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| **09:20-10:10** | **Do we know the cause of ALS-PDC?**<br>*Capsule: Pacific ALS-PDC may illuminate the causes of ALS, atypical parkinsonism and related disorders. ALS-PDC is a familial and sporadic neurodegenerative disease featured neuropathologically by a tau-dominated polyproteinopathy. Is ALS-PDC primarily a genetic disease? Others propose that environmental factors dominate the etiology of ALS-PDC.* |
| 09:20-09:30 | Host: |
| 09:30-09:45 | Yes: **Peter Spencer**, USA |
| 09:45-10:00 | No: **Helmar Lehmann**, Germany |
| 10:00-10:10 | Discussion and rebuttals |
| **10:10-10:25** | ***Coffee Break*** |
| **10:25-12:05** | **ALS THERAPY** |
| **10:25-11:15** | **Patients should set the agenda for therapeutic trials in ALS.**<br>*Capsule: There is an increasing drive of many grant-awarding bodies for applicants to demonstrate public and patient involvement. ALS patients are understandably desperate for effective therapy and frequently want to "try anything". Placebo-controlled trials may be problematic in rapidly-progressive diseases. 'Right-to-try' legislation challenges the traditional model of physician-as-expert, while unfiltered information disseminated through social media by 'expert patients' and self-appointed advocacy groups may adversely distort the research agenda.* |
| 10:25-10:35 | Host: |
| 10:35-10:50 | Yes: |
| 10:50-11:05 | No: |
| 11:05-11:15 | Discussion and rebuttals |
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| **11:15-12:05** | **The study of mice has been detrimental to developing therapy for ALS.**<br>*Capsule: ALS is a highly-selective neurodegeneration involving primarily motor possibly unique to humans. Twenty-five years since the development of the SOD1 mouse model of ALS, there are currently only two modestly disease-* |

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| | *modifying therapies for the human disorder. Have these models helped or slowed the development of therapies?* |
| 11:15-11:25 | Host: |
| 11:25-11:40 | Yes: **Peter Bede**, Ireland |
| 11:40-11:55 | No: |
| 11:55-12:05 | Discussion and rebuttals |
| **12:05-12:15** | ***Short Break*** |
| **12:15-13:15** | **Industry Symposium** |
| **13:15-14:15** | ***Lunch Break*** |
| **13:15-14:15** | **Meet the Expert** |

**SLEEP**

| **Sunday October 4, 2020** | |
|---|---|
| **14:15-15:45** | **OBSTRUCTIVE SLEEP APNEA (OSA)** |
| **14:15-15:00** | **Obstructive sleep apnea (OSA) diagnosis: A small home system is enough.**<br>*Capsule: OSA is a common disorder, especially in the group of 35-65 years of age. In this group the frequency can be large: 10-45%. The diagnosis and treatment should not be delayed. What is the best type and which is the fastest type for the right diagnosis?* |
| 14:15-14:25 | Host: **Adrian Williams**, UK |
| 14:25-14:40 | Yes: |
| 14:40-14:55 | No: **Arthur Kurvers**, The Netherlands |
| 14:55-15:00 | Discussion and rebuttals |
| **15:00-15:45** | **OSA should always be treated by CPAP.**<br>*Capsule: What are the OSA types of diagnosis? Should all types of OSA be treated the same way? In some areas all OSA patients are treated with CPAP, in other area's the treatment depends on the type of OSA and the severity. So what is the best treatment?* |
| 15:00-15:10 | Host: **Johan Verbraecken**, Belgium |
| 15:10-15:25 | Yes: **Simon Merritt**, UK |
| 15:25-15:40 | No: **Monique Vlak**, The Netherlands |
| 15:40-15:45 | Discussion and rebuttals |
| **15:45-16:00** | ***Coffee Break*** |
| **16:00-19:00** | **INSOMNIA** |
| **16:00-16:50** | **Insomnia is primarily hereditary and rarely behavioral.**<br>*Capsule: Insomnia is one of the most frequent complaints of sleep problems. Are all insomnia diagnoses the same, or are there different types? Are all cases hereditary, or is part behavioral? If there are differences, how many types are there and which is the most important group?* |
| 16:00-16:10 | Host: |
| 16:10-16:25 | Yes: |
| 16:25-16:40 | No: |
| 16:40-16:50 | Discussion and rebuttals |
| **16:50-17:40** | **Substance use and light (computers and telephones) play the most important role in causing sleep loss.**<br>*Capsule: What is the cause of lack of sleep? Is it the use of substances (i.e. alcohol, coffee, dark chocolate, green tea) and light, or is insomnia caused by hereditary factors?* |
| 16:50-17:00 | Host: **Lynn Rijsman**, The Netherlands |
| 17:00-17:15 | Yes: **Adrian Williams**, UK |
| 17:15-17:30 | No: |
| 17:30-17:40 | Discussion and rebuttals |

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| **17:40-18:30** | **Restless leg syndrome (RLS) diagnosis can be made by history alone while polysomnography (PSG) is NOT mandatory.**<br>*Capsule: RLS is a serious sleep disorder which can be diagnosed using the right questions. Others maintain that the correct diagnosis requires specific sleep measurements.* |
| 14:40-17:50 | Host: **Lynn Rijsman,** The Netherlands |
| 17:50-18:05 | Yes: **Guy Leschziner,** UK |
| 18:05-18:20 | PSG is mandatory: |
| 18:20-18:30 | Discussion and rebuttals |

**NEURODEGENERATIVE DISEASES**

| **Monday October 5, 2020** | |
|---|---|
| **07:30-08:30** | **E-Poster Presentations (Exhibition Area)** |
| **08:30-10:10** | **CAUSES OF NEURODEGENERATION** |
| **08:30-09:20** | **Can stress and anxiety cause neurodegeneration?**<br>*Capsule: In the last decades AD research has focused on possible preventable risk factors including mood and anxiety disorders studies in animals have shown that chronic stress exacerbates the deposition of proteins involved in AD in particular tau pathology. In humans, psychological stress has been associated with higher risk of AD clinical syndrome. Stress can have a damaging effect on brain health. One of the possible therapeutic target should be to mitigate the extensive negative effects of stress.* |
| 08:30-08:40 | Host: |
| 08:40-08:55 | Yes: **Luiza Spiru**, Romania |
| 08:55-09:10 | No: **Bogdan Popescu**, Romania |
| 09:10-09:20 | Discussion and rebuttals |
| **09:20-10:10** | **Do infectious agents trigger and influence neurodegeneration?**<br>*Capsule: In the last decades, infectious origin of neurodegenerative diseases, such as Parkinson's or Alzheimer's, was hypothesized. Viruses, bacteria and subtle changes in gut microbiota were incriminated. However, all these infections are still of uncertain significance in the complex cascade of pathogenic mechanisms of neurodegeneration and are denied by others. Are anti-infectious interventions worthy of clinical trial development?* |
| 09:20-09:30 | Host: **Antonio Federico**, italy |
| 09:30-09:45 | Yes: |
| 09:45-10:00 | No: **Bogdan Popescu**, Romania |
| 10:00-10:10 | Discussion and rebuttals |
| **10:10-10:25** | ***Coffee Break*** |
| **10:25-12:55** | **DIAGNOSIS** |
| **10:25-11:15** | **Development of precise preclinical diagnosis of neurodegenerative diseases – illusion or reality?**<br>*Capsule: AD is the only example of preclinical diagnosis in the neurodegenerative domain with enough studies, yet the role of additional pathways and neuropathology, Lewy bodies, TDP-43, vascular lesions and severity of AD are critical for understanding pathogenesis.* |
| 10:25-10:35 | Host: |
| 10:35-10:50 | Illusion: |
| 10:50-11:05 | Reality: |
| 11:05-11:15 | Discussion and rebuttals |

**REHABILITATION**

| **Monday October 5, 2020** | |
|---|---|
| **07:30-08:30** | **E-Poster Presentations (Exhibition Area)** |
| **08:30-10:10** | **TREATMENT OF PROGRESSIVE NEURODEGENERATIVE DISEASES** |
| **08:30-09:20** | **Neurorehabilitation in progressive neurological disorders.** |

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| | *Capsule: Neurorehabilitation program improve functions and wellbeing of people with progressive neurological disorders at least for a period of time. Should we prescribe the same "maintenance comprehensive therapy" even in the advanced stages of the deterioration, or should we apply an "adapted reduced one" ?* |
| 08:30-08:40 | Host: **Ales Praznikar**, UK |
| 08:40-08:55 | A short period is enough: |
| 08:55-09:10 | Continue indefinitely: |
| 09:10-09:20 | Discussion and rebuttals |
| **09:20-10:10** | **Is physical therapy useful in PD?** |
| | *Capsule: Comprehensive treatment approach toward patients with PD includes physical therapy. There is some evidence that intensive physical therapy is helpful. However, this evidence is mainly subjective and no class A data exists. Is this enough to recommend physical therapy in all PD patients?* |
| 09:20-09:30 | Host: |
| 09:30-09:45 | Yes: |
| 09:45-10:00 | No: **Ovidiu Bajenaru**, Romania |
| 10:00-10:10 | Discussion and rebuttals |
| **10:10-10:25** | ***Coffee Break*** |
| **10:25-12:55** | **REHABILITATION** |
| **10:25-11:15** | **Upper limb recovery in stroke patients – standalone or combined with pharmacological support?** |
| | *Capsule: Experience in neurorehabilitation has shown that the pattern of upper limb functional recovery after acute ischemic stroke can be modified by intensive task-oriented, learning-dependent recovery strategies. Nevertheless, the overall recovery potential of the individual is mainly influenced by intrinsic mechanisms. Are physical therapy and early mobilization enough to stimulate endogenous neurorecovery pathways? Can pharmacological intervention enhance upper limb neurorehabilitation, contributing to an improved outcome?* |
| 10:25-10:35 | Host: |
| 10:35-10:50 | With support: |
| 10:50-11:05 | Standalone: **Ales Praznikar**, UK |
| 11:05-11:15 | Discussion and rebuttals |
| **11:15-12:05** | **Is neuroimaging helpful during rehabilitation from stroke?** |
| | *Capsule: When patients with cerebral stroke are admitted to the departments of rehabilitation medicine, the general approach is a comprehensive one by a multi-disciplinary assessment and the "tailor made" treatment program tries to fit the proper one for every patient. Along the process, various neuroimaging are carried out. Neurologists, rehabilitation physicians, neuro-psychologists and neuro-radiologists, try to compare the radiological findings with the clinical ones. But, in fact, the rehab process continues in spite of frequent discrepancies. Is neuroimaging really helpful?* |
| 11:15-11:25 | Host: |
| 11:25-11:40 | Yes: |
| 11:40-11:55 | No: |
| 11:55-12:05 | Discussion and rebuttals |