PLENARY

Thursday, March 26, 2020	
09:00-11:00	**SESSION 1 | OPENING SESSION**
09:00-09:30	Welcome remarks **Amos Korczyn,** Israel and **Anthony Schapira**, UK
09:30-10:00	PD from mitochondria to lysosomes and on to therapy **Anthony Schapira**, UK
10:00-10:30	
10:30-11:00	Should cannabinoids be legally available to the public? **Peter Feldschreiber**, UK
11:00-11:30	*Coffee Break*
11:30-13:00	**SESSION 2 | PLENARY LECTURES**
11:30-12:00	Neurostimulation and its potential to improve learning and cognition in health and disease **Roi Cohen Kadosh**, UK
12:00-12:30	Functional neurological disorders **Stoyan Popkirov**, Germany
12:30-13:00	Pathological impulsivity - a neurobehavioural phenomenon **Valerie Voon**, UK
13:00-13:45	*Lunch Break*
13:45-15:15	**SESSION 3 | PLENARY LECTURES**
13:45-14:15	GAD antibodies spectrum disorder **Marinos Dalakas**, USA
14:15-14:45	MS is one disease; why definitions matter to patients and how we treat them **Gavin Giovannoni**, UK
14:45-15:15	Digital health technologies in clinical care and clinical trials **Jesse Cedarbaum**, USA
15:15-15:30	*Coffee Break*
15:30-18:20	**SESSION 4 | PLENARY LECTURES**
15:30-16:00	**TBD**
16:00-16:30	The two in one brain circulations **Vladimir Hachinski**, Canada
16:30-17:00	Emerging neuropathological comorbidities in aging - LATE, ARTAG and CTE **Lea Grinberg**, USA/Brazil
17:00-18:00	**GWAS is likely to contribute significantly to Alzheimer's disease (AD) patients care.** *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 find a cure for AD?*
17:00-17:10	Host: **Peter Whitehouse**, USA
17:10-17:25	Yes: **John Hardy**, UK
17:25-17:40	No: **Amos Korczyn**, Israel
17:40-17:50	Discussions and rebuttals
17:50-18:20	Thomas Willis and the first decade of the brain **Alastair Compston**, UK
18:20	**Welcome Reception**

Friday, March 27, 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?** *Capsule: The 2017 revisions of the McDonald criteria for the diagnosis of MS were mainly designed to facilitate an earlier MS diagnosis thus marginalizing 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.*
08:30-08:40	Host: **Ralf Linker**, Germany
08:40-08:55	Yes: **Brian Weinshenker**, USA
08:55-09:10	No: **Christopher Hawkes**, UK
09:10-09:20	Discussion and rebuttals
09:20-10:10	**Does OCT make VEP redundant?** *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: **Hadas Stiebel-Kalish**, Israel
09:45-10:00	No: **Mario Habek**, Croatia
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 subjected to clinical trials even with poor scientific support?** *Capsule: Over the past three decades, numerous drugs were approved for multiple sclerosis (MS), but in many patients the disease is not fully controlled. In this session, the debaters will outline the pros and cons of using interventions based on limited 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: **Richard Nicholas,** UK
10:50-11:05	No: **Konrad Rejdak**, Poland
11:05-11:15	Discussion and rebuttals
11:15-12:05	**Newly diagnosed MS patients should be started on aggressive therapy.** *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: **Jera Kruja**, Albania
11:25-11:40	Yes: **Ron Milo**, Israel
11:40-11:55	No: **Uros Rot**, Slovenia
11:55-12:05	Discussion and rebuttals
12:05-12:15	***Technical 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	**DISEASE COURSE**
14:15-15:00	**Are MS patients at increased risk for developing cancer?** *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. On the other hand, some register and*

	large cohort studies 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: **Ali Manouchehrinia**, Sweden
14:40-14:55	No: **Melinda Magyari**, Denmark
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.** *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 inflammation disease of the brain with secondary degeneration?*
15:00-15:10	Host: **Bart van Wijmeersch**, Belgium
15:10-15:25	Yes: **Antonio Scalfari**, UK
15:25-15:40	No: **Bianca Weinstock-Guttman**, USA
15:40-15:45	Discussion and rebuttals
15:45-16:00	***Coffee Break***
16:00-18:45	**COGNITION IN MS**
16:00-16:55	**In MS patients with significant cognitive decline, drug treatment should be modified.** *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?*
16:00-16:10	Host: **Laszlo Vecsei**, Hungary
16:10-16:25	Switch to a new agent: **Ron Milo**, Israel
16:25-16:40	Not so fast: **Amos Korczyn**, Israel
16:40-16:55	Discussion and rebuttals
16:55-17:50	**Cognitive decline is sufficient to define transition to secondary progressive multiple sclerosis (SPMS).** *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 many years. Is cognitive decline sufficient to define this change?.*
16:55-17:05	Host: **Dimitrios Karussis**, Israel
17:05-17:20	Yes: **Klaus Schmierer**, UK
17:20-17:35	No: **Thomas Berger**, Austria
17:35-17:50	Discussion and rebuttals
17:50-18:45	**We are well enough equipped to identify fake news in MS therapy before it can cause harm.** *Capsule: Fake news is 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*
17:50-18:00	Host: **Nikos Evangelou**, UK
18:00-18:15	Yes: **Klaus Schmierer**, UK
18:15-18:30	No: **Radu Tanasescu**, UK
18:30-18:45	Discussion and rebuttals

STROKE

Friday, March 27, 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?** *Capsule: EUS 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 required for treatment decision in patients arriving more than six hours after stroke onset or in those with sleep onset strokes?*
08:30-08:40	Host: **Joanna Wojczal**, Poland
08:40-08:55	Yes: **Krassen Nedeltchev**, Switzerland
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?** *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 negative DWI imaging really exist?*
09:20-09:30	Host: **Adrian Perry-Jones**, UK
09:30-09:45	Yes: **Jonathan Streifler**, Israel
09:45-10:00	No: **Krassen Nedeltchev**, Switzerland
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: Patients with embolic stroke of unknown source (ESUS) 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: **Natan Bornstein**, Israel
10:35-10:50	Yes: **David Spence**, Canada
10:50-11:05	No: **Jonathan Streifler**, Israel
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 in patients which nonvalvular atrial fibrillation are associated with left atrial thrombi and left atrial appendage closure may be suitable alterative to chronic anticoagulation.*
11:15-11:25	Host: **George Chrysant**, USA
11:25-11:40	Yes: **Roni Eichel**, Israel
11:40-11:55	No: **Michael Glikson**, Israel
11:55-12:05	Discussion and rebuttals
12:05-12:15	***Technical 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 STROKE MANAGEMENT**
14:15-15:00	**Mobile stroke units (MSU) are useful and cost effective.** *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: **Krassen Nedeltchev**, Switzerland
14:55-15:00	Discussion and rebuttals

15:00-15:45	**Does the main benefit of AIS 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: **Agnieszka Słowik**, Poland
15:10-15:25	tPA: **Gary Ford**, UK
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 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: **Ashfaq Shuaib**, Canada
16:10-16:25	Yes: **David Spence**, Canada
16:25-16:40	No: **Vida Demarin**, Croatia
16:40-16:45	Discussion and rebuttals
16:45-17:30	**Should symptomatic extracranial vertebral artery stenosis be stented?** *Capsule: Stenosis in the vertebro-basilar system accounts for about a 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 ASA and clopidogrel?** *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. Is there sufficient evidence to recommend long-term dual antiplatelet therapy for all patients with TIA?*
17:30-17:40	Host: **Agnieszka Słowik**, Poland
17:40-17:55	Yes: **Jorge Celis**, Colombia
17:55-18:10	No: **David Spence**, Canada
18:10-18:15	Discussion and rebuttals
18:15-19:00	**In the presence of cerebral microbleeds (CMBs), antithombotic therapy should be avoided.** *Capsule: The presence of microbleeds (detected only with MRI) may increase the 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: **David Werring**, UK
18:25-18:40	Yes:
18:40-18:55	No: **Laszlo Csiba**, Hungary
18:55-19:00	Discussion and rebuttals

Friday, March 27, 2020	
07:30-08:30	**E-Poster Presentations (Exhibition Area)**
08:30-10:10	**PRECLINICAL AND EARLY ALZHEIMER'S DISEASE (AD)**
08:30-09:20	**Is subjective cognitive impairment itself a prelude to dementia?** *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 and the associated clinical decline. 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?*
08:30-08:40	Host: **David Knopman**, USA
08:40-08:55	Yes: **Babak Tousi**, USA
08:55-09:10	No: **Panteleimon Giannakopoulos**, Switzerland
09:10-09:20	Discussion and rebuttals
09:20-10:10	**Is cognitive reserve a buzzword lacking scientific value?** *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.** *Capsule: AD is characterized by cognitive deterioration, but non-cognitive behavioral symptoms are also frequent and often associated with more suffering than cognitive and functional decline. Treatment of those symptoms may be difficult and challenging, and there is insufficient evidence to support treatment decisions. Often a combination of different non-pharmacological approaches precedes drug treatment. This debate will focus on the question whether the improved understanding of AD pathology has improved patient care and treatment, including non-cognitive symptoms.*
10:25-10:35	Host: **Ruth Itzhaki**, UK
10:35-10:50	Yes: **Judith Aharon**, Israel
10:50-11:05	No: **Sagrario Manzano**, Spain
11:05-11:15	Discussion and rebuttals
11:15-12:05	**Vascular lesions contribute to AD pathology.** *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, dyslipidaemia and adiposity are all linked to AD. However, it is unclear whether these factors contribute to or promote AD pathology itself. This debate will address whether vascular changes are just coincident with AD type of pathology or part and parcel of AD.*
11:15-11:25	Host: **Nick Fox**, UK
11:25-11:40	Yes: **Rajesh Kalaria**, UK
11:40-11:55	No: **David Knopman**, USA
11:55-12:05	Discussion and rebuttals

12:05-12:15	***Technical 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?** *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 or if it mitigates enhanced risk for dementia via other factors is less well understood. This debate will discuss if physical activity protects cognitive function and whether we know enough about the phenomenon to design effective interventions.*
14:15-14:25	Host: **Miia Kivipelto**, Sweden/UK
14:25-14:40	Yes: **Catherine Robb**, UK
14:40-14:55	No: **Naji Tabet**, UK
14:55-15:00	Discussion and rebuttals
15:00-15:45	**Is deafness a causative risk factor to dementia?** *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 but 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: **Lev Kruglov**, Russia
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?** *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: **Paul Edison**, UK
16:25-16:40	No: **Mee Park**, South Korea
16:40-16:45	Discussion and rebuttals
16:45-17:30	**Will big data help us to find cure for dementia?** *Capsule: In recent years there has been a surge towards big data approaches in AD, following a general trend in other disciplines in medicine and beyond. 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 itself. 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: **John Gallacher**, UK
17:10-17:25	No: **Eugen Tarnow**, USA
17:25-17:30	Discussion and rebuttals
17:30-19:00	**FRONTOTEMPORAL DEMENTIA**
17:30-18:15	**The term Frontotemporal Dementia (FTD) is no longer of value.** *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: **Janine Diehl-Schmid**, Germany
17:40-17:55	Yes: **Eugen Tarnow**, USA
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.** *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. Mutations in other genes have also been described but are much rarer than the three mentioned above. 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: **John Hardy**, UK
18:25-18:40	Yes: **Lea Grinberg**, Brazil/USA
18:40-18:55	No:
18:55-19:00	Discussion and rebuttals

HEADACHE

Friday, March 27, 2020	
07:30-08:30	**E-Poster Presentations (Exhibition Area)**
08:30-10:10	**SLEEP, HORMONES AND MEDICATION OVERUSE HEADACHE**
08:30-09:20	**Estrogen containing contraceptives are safe in women with migraine with aura.** *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. There is a lack of good quality studies assessing risk of stroke associated with low dose estrogen use in women with migraine.*
08:30-08:40	Host: **Pooja Dassan**, UK
08:40-08:55	Yes: **Anne MacGregor**, UK
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.** *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: **Jack Schim,** USA
09:30-09:45	Yes: **Bojana Zvan**, Slovenia
09:45-10:00	No: **Oved Daniel**, Israel
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.** *Capsule: CGRP is known to be widely distributed in the central and peripheral nervous system.The exact site of action of the monoclonal antibodies to CGRP or its receptor and small molecule CGRP receptor blockers 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 well known enough for physicians to recommend them for long-term use.** *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 in the future. CGRP is also involved in many other processes such as bone and wound healing as well as cardiovascular homeostasis.*
11:10-11:20	Host: **Giorgio Lambru**, UK
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?** **Christopher Gottschalk**, USA
12:15-13:15	**Industry Supported Symposium**
13:15-14:15	***Lunch Break***
13:15-14:15	**Meet the Expert**
14:15-15:45	**ATYPICAL MIGRAINE**
14:15-15:00	**Head injury can precipitate the onset of migraine**. *Capsule: Post-traumatic headache may occur in several phenotypes. Can a patient develop migraine with or without aura due to head trauma?*
14:15-14:25	Host: **Manjit Matharu**, UK
14:25-14:40	Yes: **Oved Daniel**, Israel
14:40-14:55	No: **Mark Braschinsky**, Estonia
14:55-15:00	Discussion and rebuttals
15:00-15:45	**Vestibular migraine – does it exist?** *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: **Min Kyung Chu**, South Korea
15:10-15:25	Yes: **Christian Lampl**, Austria
15:25-15:40	No: **Jack Schim**, USA
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	**Acupuncture is as effective as migraine preventive medications, with fewer unwanted adverse effects.** *Capsule: In recent years the evidence base for acupuncture as a preventive treatment for migraine has grown considerably due to the publication of several large trials of high quality; however the results are still questioned and not easy to interpret.*
16:00-16:10	Host: **Jose Miguel Lainez**, Spain
16:10-16:25	Yes: **Min Kyung Chu**, South Korea
16:25-16:40	No: **Surat Tanprawate**, Thailand
16:40-16:50	Discussion and rebuttals
16:50-17:40	**Headache devices will replace medications for the acute and preventive treatment of migraine and cluster headache.** *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. What is the evidence?*

16:50-17:00	Host: **Anna Anrdreou**, UK
17:00-17:15	Yes: **Jose Miguel Lainez**, Spain
17:15-17:30	No: **Giorgio Lambru**, UK
17:30-17:40	Discussion and rebuttals
17:40-18:30	**Mushrooms are a good treatment for chronic cluster headache.** *Capsule: Psilocybin, lysergic acid diethylamide, 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?*
17:40-17:50	Host: **Dimos Mitsikostas**, Greece
17:50-18:05	Yes: **Brian E. McGeeney**, USA
18:05-18:20	No: **Randall Weeks**, USA
18:20-18:30	Discussion and rebuttals
18:30-19:00	The changing face of medication-overuse headache **Alan Rapoport**, USA

PARKINSON'S DISEASE

Saturday, March 28, 2020	
07:30-08:30	**E-Poster Presentations (Exhibition Area)**
08:30-10:10	**CONTROVERSIES IN ADVANCED PARKINSON'S DISEASE (PD)**
08:30-09:20	**When is the right time to refer PD patients for deep brain stimulation (DBS)?** *Capsule: DBS is effective in treating of medication-refractory symptoms (motor and non-motor) and improving 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, short and long-term safety profile and device design require us to re-evaluate the right time for DBS to earlier stages of the disease than for which it is currently used and to confer long-term symptomatic benefit for patients*
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: **Fiona Gupta**, USA
09:30-09:45	Yes: **Abdelhamid Benazzouz**, France
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 not required.** *Capsule: PD is primarily a motor disorder, yet non-motor symptoms become more widely recognized. How important are these NMS in the clinical assessment of the patient?*
10:25-10:35	Host: **Irene Litvan**, USA
10:35-10:50	Yes: **Kailash Bhatia**, London
10:50-11:05	No: **Pedro Garcia Ruiz**, Spain
11:05-11:15	Discussion and rebuttals

11:15-12:05	**Should untroubling dyskinesia be treated?** *Capsule: Dyskinesia occurs frequently in advanced PD patients treated with dopaminergic medications. The Dyskinesia frequently is troubling to spouses or caregivers more than to the patient themselves, particularly in mild cases. Should these patients still be treated?*
11:15-11:25	Host: **Daniel Kremens**, USA
11:25-11:40	Yes: **Rajesh Pahwa**, USA
11:40-11:55	No: **Ray Chaudhuri**, UK
11:55-12:05	Discussion and rebuttals
12:05-12:15	***Technical 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	**GENES AND ENVIRONMENT**
14:15-15:00	**Should PD patients carrying GBA mutations be treated differently from gene negative?**
14:15-14:25	Host: **Vladimira Vuletic**, Croatia
14:25-14:40	Yes: **Anthony Schapira**, UK
14:40-14:55	No: **Rajesh Pahwa**, USA
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 which characterize idiopathic PD is not fully understood. Although several genes were found responsible for the development of PD, the causative agents for a great percent of cases remain unclear. Various factors were incriminated to increase the risk of PD development. But their interactions with genetic factors are unknown role in PD pathogenesis.*
15:00-15:10	Host: **Alastair Noyce**, UK
15:10-15:25	Yes: **Cristian Falup-Pecurariu**, Romania
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?** *Capsule: A change in the content of α-synuclein (α-SN) in CSF is considered as a promising biomarker of PD. Indeed, the total α-SN content in CSF decreases, and the fractions of phosphorylated and oligomeric α-SN increase in PD compared to aging controls. However, all attempts to use α-SN as a biomarker for differential diagnosis or prognosis were unsuccessful. It is hoped that α-SN in CSF can be used as a biomarker for the differential diagnosis of PD, but only in combination with biomarkers of other diseases.*
16:00-16:10	Host: **Michael Ugrumov**, Russia
16:10-16:25	Yes: **Georgia Xiromerisiou**, Greece
16:25-16:40	No: **Mark F. Lew**, USA
16:40-16:50	Discussion and rebuttals
16:50-17:40	**Is continuous dopaminergic stimulation better strategy than pulsatile in every patient?**
16:50-17:00	Host: **Fernando Pagan**, USA
17:00-17:15	Yes: **Stuart Isaacson**, USA
17:15-17:30	No: **Tatyana Simuni**, USA
17:30-17:40	Discussion and rebuttals
17:40-18:10	The personalized Parkinson's

	Ray Chaudhuri, UK
18:10-18:15	Discussion
18:15-19:00	**CLINICAL PATHOLOGICAL CONFERENCE (CPC)**
Moderator:	**Tom Warner**, UK
18:15-19:00	*Tamas Revesz* –CBD case with a PSP clinical phenotype (CBD-PSPS) **Helen Ling**, UK

EPILEPSY

Saturday, March 28, 2020	
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.** *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?*
08:30-08:40	Host: **Mark Richardson**, UK
08:40-08:55	Yes: **Antonio Gil-Nagel**, Spain
08:55-09:10	No: **Dana Ekstein**, 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.** *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: **Ivan Rektor**, Czech Republic
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 in psychogenic seizures. 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: **Christian Bien**, Germany
11:55-12:05	Discussion and rebuttals
12:05-12:15	***Technical 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**
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 antiepileptic drugs, 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**
16:00-16:50	**Should surgery to be offered to patients after failure of two AED?** *Capsule: Epidemiological studies suggest that drug failure is quite likely once two agents have failed to control seizures. The literature contains numerous reports of response to drug therapy in patients formerly considered drug resistant. Are the ILAE guidelines supported by the evidence?*
16:00-16:10	Host: **Manjari Tripathi**, India
16:10-16:25	Yes: **Zeljka Petelin Gadže**, Croatia
16:25-16:40	No: **Ettore Beghi**, Italy
16:40-16:50	Discussion and rebuttals
16:50-17:40	**The newer AED are more effective than the established ones.** *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 in children and adults. Have they improved overall outcomes in terms of seizure freedom and so proved value for money?*
16:50-17:00	Host: **Michael Sperling**, USA
17:00-17:15	Yes: **Andreas Schulz-Bonhage**, Germany
17:15-17:30	No: **Martin Brodie**, UK
17:30-17:40	Discussion and rebuttals
17:40-19:00	**Epilepsy Cases –** Challenging diagnostic and management cases will be presented to the faculty and audience for discussion. A lively debate is anticipated for each case. **Michael Sperling**, USA

DEMENTIA SATELLITE – OVERCOMING THE IMPASSE IN DEMENTIA PREVENTION

Saturday, March 28, 2020	
07:30-08:30	**E-Poster Presentations (Exhibition Area)**
08:30-09:00	The complex reality **Amos Korczyn**, Israel

09:00-09:10	Discussion
09:10-09:25	Introduction from the Alzheimer's Association
09:25-09:55	Animal models of Alzheimer's disease **Andrea Tanner**, USA
09:55-10:10	Discussion
10:10-10:25	***Coffee Break***
10:25-10:55	Are environmental interventions (optimal CV control, diet, physical activity, *etc*.) likely to change the frequency and by how much? **Kristine Yaffe**, UK
10:55-11:20	Discussion
11:20-11:50	What can pathology contribute to our understanding, given that autopsies come very late in the disease course and show mixed pathology in most cases? **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**
14:15-14:45	Are amyloid and tau dead horses in sporadic, late onset AD, given the disappointments with interventions that were successful in eliminating amyloid yet without clinical benefit? **David Knopman**, USA
14:45-15:00	Discussion
15:00-15:30	To futility or not – when and how should you apply futility analysis be applied **Rema Raman**, USA
15:30-15:45	Discussion
15:45-16:00	***Coffee Break***
16:00-16:30	Is APOE4 a potential treatment target, given that we do not understand its mechanism? **Daniel Michaelson**, Israel
16:30-16:45	Discussion
16:45-17:15	Is neuroinflammation a useful potential therapeutic target? **Michael Heneka**, Germany
17:15-17:30	Discussion
17:30-18:00	Fear and loathing in recent Alzheimer trials: Aducanumab, albumin/IVIG exchange, and oligomanurate **Lon Schneider**, USA
18:00-18:15	Discussion
18:15-18:45	The need for multiple targets, outcomes, and approaches **Vladimir Hachinski**, Canada
18:45-19:00	Discussion

NEURO-IMMUNOLOGY

Saturday, March 28, 2020	
07:30-08:30	**E-Poster Presentations (Exhibition Area)**
08:30-10:10	**PANDAS AND CIDP**
08:30-09:20	**PANDAS/PANS is a clinically distinct entity and needs early and prompt immunotherapy.** *Capsule: The nosology of pediatric acute neuropsychiatric syndromes like PANAS and PANS remains controversial, and the evidence for immunopathology and in particular autoimmunity driven by Streptococcal infection remains controversial. Is this a distinct disorder and is prompt immunotherapy indicated for treatment?*
08:30-08:40	Host: **Alicja Kalinowska**, Poland

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	**Treatment of CIDP - immunoglobulins or steroids first?** *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?*
09:20-09:30	Host: **Joab Chapman**, Israel
09:30-09:45	Immunoglobulins: **Marinos Dalakas**, USA
09:45-10:00	Steroids: **Eduardo Nobile-Orazio**, Italy
10:00-10:10	Discussion and rebuttals
10:10-10:25	***Coffee Break***
10:25-12:05	**NEUROMYELITIS OPTICA SPECTRUM DISORDER**
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.** *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 widely-used immunotherapies that. Are widely regarded as effective and are less expensive?*
10:25-10:35	Host: **Brian Weinshenker**, USA
10:35-10:50	Yes: **Friedemann Paul**, Germany
10:50-11:05	No: **Andrzej Glabinski**, Poland
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.** *Capsule: Recent retrospective experience suggests that concomitant or first line treatment with plasma exchange may be superior to first line 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: **Ingo Kleiter**, Germany
11:40-11:55	No: **M Isabel Leite**, UK
11:55-12:05	Discussion and rebuttals
12:05-12:15	***Technical Break***
12:15-13:15	**Industry Symposium**
13:15-14:15	***Lunch Break***
13:15-14:15	**Meet the Expert**
14:15-15:00	**The 2015 International Panel criteria for NMOSD are outdated and should be replaced with a diagnostic classification primarily based on autoantibody status rather than clinical presentation (e.g. AQP4 disease, MOG disease).** *Capsule: We now know the molecular target of the autoimmune insult in the majority of patients with NMOSD, and we can use a molecular classification based on the target of the antibody 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: **Jacek Losy**, Poland
14:25-14:40	Yes: **Angela Vincent**, UK
14:40-14:55	No: **Ingo Kleiter**, Germany
14:55-15:00	Discussion and rebuttals
15:00-15:45	**Is narcolepsy an autoimmune disorder which should be treated as such?**
15:00-15:10	Host: **Friedemann Paul**, Germany
15:10-15:25	Yes: **Jack Losy**, Poland
15:25-15:40	No: **Angela Vincent**, UK
15:40-15:45	Discussion and rebuttals
15:45-16:00	***Coffee Break***

Saturday, March 28, 2020	
07:30-08:30	**E-Poster Presentations (Exhibition Area)**
08:30-09:20	**ALS should not be considered a neuromuscular disorder.** *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, which 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: **Michael van Es**, The Netherlands
08:55-09:10	No: **Monica Povedano Panades**, Spain
09:10-09:20	Discussion and rebuttals
09:20-10:10	**Solving the etiology of Western Pacific ALS-PDC may illuminate understanding of the causes of ALS, atypical parkinsonism and related disorders elsewhere. Do we know the environmental cause of ALS-PDC?** *Capsule: Western Pacific amyotrophic lateral sclerosis and parkinsonism-dementia complex (ALS-PDC) is a familial and sporadic neurodegenerative disease featured neuropathologically by a tau-dominated polyproteinopathy. Over the past half-century in the hyperendemic communities of Guam, and Kii Peninsula, disease incidence has progressively declined. Is ALS-PDC primarily a genetic disease.. Similarly, on Guam, it is suggested that family histories with multi-incident pedigrees (ALS, atypical parkinsonism, dementia and PDC) are compatible with genetic transmission of each syndrome. Others propose that environmental factors dominate the etiology of ALS-PDC, the chemical identity of which is relevant to related neurodegenerative disorders (e.g., ALS, progressive supranuclear palsy.*
09:20-09:30	Host: **Albert Ludolph**, Germany
09:30-09:45	Yes: **Peter Spencer**, USA
09:45-10:00	No:
10:00-10:10	Discussion and rebuttals
10:10-10:25	***Coffee Break***
10:25-11:15	**Patients should set the agenda for therapeutic trials in ALS.** *Capsule: Research being carried out 'with' or 'by' members of the public rather than 'to', 'about' or 'for' them, underpins an increasing drive of many grant-awarding bodies for applicants to demonstrate public and patient involvement (PPI). ALS patients are understandably desperate for more 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: **Albert Ludolph**, Germany
10:35-10:50	Yes: **Paul Wicks**, USA
10:50-11:05	No:
11:05-11:15	Discussion and rebuttals
11:15-12:05	**The study of mice has been detrimental to developing therapy for ALS.** *Capsule: ALS is a highly-selective neurodegeneration involving motor and extra-motor neuronal networks possibly unique to humans. Twenty-five years since the development of the SOD1 mouse model of ALS, there are currently two only modestly disease-modifying therapies for the human disorder. Transgenic over-expression models may not capture the cellular ageing processes in human neurodegenerative disorders, but rodents offer systems-level readout not currently possible using iPSC-derived models.*
11:15-11:25	Host:
11:25-11:40	Yes: **Peter Bede**, Ireland
11:40-11:55	No: **Elizabeth Fisher**, UK
11:55-12:05	Discussion and rebuttals
12:05-12:15	***Technical Break***
12:15-13:15	**Industry Symposium**
13:15-14:15	***Lunch Break***
13:15-14:15	**Meet the Expert**

SLEEP

Saturday, March 28, 2020	
14:15-15:00	**OSA diagnosis with small home systems (such as WP) is enough.**
14:15-14:25	Host: **Adrian Williams**, UK
14:25-14:40	Yes: **Hans Hamburger**, The Netherlands
14:40-14:55	No: **Arthur Kurvers**, The Netherlands
14:55-15:00	Discussion and rebuttals
15:00-15:45	**OSA treatment should always be done by CPAP.**
15:00-15:10	Host: **Johannes Verbraecken**, Belgium
15:10-15:25	Yes:
15:25-15:40	No:
15:40-15:45	Discussion and rebuttals
15:45-16:00	***Coffee Break***
16:00-16:50	**Insomnia is always hereditary and rarely behavioral.**
16:50-17:40	**Substance use (like alcohol, coffee, dark chocolates, green tea) and light (computers and telephones) play the most important role in causing sleep loss.**
16:50-17:00	Host:
17:00-17:15	Yes: **Adrian Williams**, UK
17:15-17:30	No: **Laurien Teunissen**, The Netherlands
17:30-17:40	Discussion and rebuttals
17:40-18:30	**Insomnia can be cured by App's only.**

NEURODEGENERATION

Sunday, March 29, 2020	
07:30-08:30	**E-Poster Presentations (Exhibition Area)**
08:30-09:20	**Can stress and anxiety cause neurodegeneration?**
08:30-08:40	Host: **Giancarlo Logroscino**, Italy
08:40-08:55	Yes:
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?**
09:20-09:30	Host: **Antonio Federico**, Italy
09:30-09:45	Yes: **Peter Jenner,** UK
09:45-10:00	No: **Bogdan Popescu**, Romania
10:00-10:10	Discussion and rebuttals
10:10-10:25	***Coffee Break***
10:25-11:15	**Development of precise preclinical diagnosis of neurodegenerative diseases – illusion or reality?**
10:25-10:35	Host:
10:35-10:50	Illusion: **Giancarlo Logroscino**, Italy
10:50-11:05	Reality: **Tamas Revesz**, UK
11:05-11:15	Discussion and rebuttals
11:15-12:05	**Can physical activity delay neurodegeneration?**

12:05-12:55	**Does metabolic imaging add to MRI in defining diagnosis in patients with neuro-degenerative diseases?**

REHABILITATION

Sunday, March 29, 2020	
07:30-08:30	**E-Poster Presentations (Exhibition Area)**
08:30-09:20	**Neurorehabilitation in progressive neurological disorders?**
09:20-10:10	**Is physical therapy useful in PD?**
10:10-10:25	***Coffee Break***
10:25-11:15	**Upper limb recovery in stroke patients – standalone or combined with pharmacological support.**
11:15-12:05	**Is neuro imaging helpful during rehabilitation from stroke.**

MOVEMENT DISORDER

Sunday, March 29, 2020	
07:30-08:30	**E-Poster Presentations (Exhibition Area)**
08:30-09:20	**Is there a role for stereotactic ablation in movement disorders in the DBS era?**
08:30-08:40	Host: **Ilana Schlesinger**, Israel
08:40-08:55	Yes: **John Duda**, USA
08:55-09:10	No: **Abdelhamid Benazzouz**, France
09:10-09:20	Discussion and rebuttals
09:20-10:10	**DBS in tardive dyskinesia?** *Capsule: Treatment options for tardive dyskinesias include pharmacological interventions (oral medications and Botulinum toxin injections), but the effects are limited and new pharmacological forms of tetrabenazine (eg valbenazine) are expensive. Other limitations may be the result of side effects of medications (e.g. depression or parkinsonism after tetrabenazine) and very complex form of abnormal movements (e.g. oromandibular or other segmental dystonia), difficult to treat with local injections of Botulinum toxin. Therefore, however the evidence is limited DBS may offer a good treatment option for refractory and most severe cases.*
09:20-09:30	Host: **Laxman Bahroo**, USA
09:30-09:45	Yes: **Jaroslaw Slawek**, Poland
09:45-10:00	No:
10:00-10:10	Discussion and rebuttals
10:10-10:25	***Coffee Break***
10:25-11:15	**RLS diagnosis can be made by history alone, PSG is NOT mandatory.**
10:25-10:35	Host: **Lynn Rijsman,** The Netherlands
10:35-10:50	Yes: **Guy Leschziner,** UK
10:50-11:05	PSG is mandatory: **Cristian Falup-Pecurariu**, Romania
11:05-11:15	Discussion and rebuttals
11:15-12:05	**PET Imaging is an expensive tool with poor external validity and not relevant for future biomarker projects.**
11:15-11:25	Host: **John Duda**, USA
11:25-11:40	Yes: **Jesse Cedarbaum**, USA
11:40-11:55	No: **Nikhil Sharma**, UK
11:55-12:05	Discussion and rebuttals

12:05-12:55	**Is there a role for neurosurgery in refractory Tourette syndrome?**
12:05-12:15	Host: **Nestor Galvez**, USA
12:15-12:30	Yes: **Patricia Limousin**, UK
12:30-12:45	No:
12:45-12:55	Discussion and rebuttals