CONy 2022 Virtual Congress Scientific Program

Program times refer to Central European Time (CET)

THURSDAY, MARCH 24, 2022		
14:00-19:20	**OPENING PLENARY SESSION**	**HALL A**
Chair:	**Natan Bornstein**, Israel	
14:00-14:10	Welcome remarks by CONy Co-Chairs **Anthony Schapira**, UK, **Natan Bornstein**, Israel	
14:10-14:20	Opening remarks by CONy President **Amos D. Korczyn**, Israel	
14:20-14:50	GBA-related Parkinson's disease **Anthony Schapira**, UK	
14:50-15:20	Cannabis and Covid 19: Is the benefit worth the risk **Peter Feldschreiber**, UK	
15:20-15:50	Functional neurological disorders **Mark Hallett,** USA	
15:50-16:20	Neurostimulation to improve cognition in health and disease **Roi Cohen Kadosh**, UK	
16:20-16:50	Impulse Control Disorders in PD **Valerie Voon**, UK	
16:50-17:20	The impact of mental trauma on cognition in older refugees in the middle east **Tala Al Rousan**, USA	
17:20:17:50	Theory of Mind **Ray Dolan**, UK	
17:50-18:20	Climate change and neurology **Sanjay Sisodiya**, UK	
18:20-18:50	Selective vulnerability of neurons in neurodegenerative diseases **Lea Grinberg**, USA/Brazil	
18:50-19:40	**Is cognitive reserve a useful concept?**	
	Capsule: Emergence of dementia occurs in different individuals at different ages. The reasons for the heterogeneity is unknown, but it has been hypothesized that this is due to "cognitive reserve", which protects the brain from either neurodegeneration or the expression of neuronal loss. This debate will explore whether the introduction of the terms cognitive reserve is useful.	
18:50-19:00	Introduction and Pre-Debate Voting	
19:00-19:15	YES: **Eider Arenaza-Urquijo**, Spain	
19:15-19:30	NO: **Amos D. Korczyn**, Israel	
19:30-19:40	Discussion, Rebuttals and Post-Debate Voting	

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FRIDAY, MARCH 25, 2022		
14:00-16:30	**HEADACHE**	**HALL A**
Chair:	**Dimos Mitsikostas**, Greece	
14:00-14:50	**Do white matter hyperintensities in MRI of migraine patients have clinical significance?**	
	Capsule: Diffuse small white matter changes may be present in a proportion of patients with migraine. Whether these changes are more frequent in migraineurs than in non-migraineurs remains controversial. It has been hypothesized that these lesions may be related to migraine progression into a chronic state affecting the sensory processing substantially. Are there clinical implications?	
14:00-14:10	Introduction and Pre-Debate Voting	
14:10-14:25	YES: **Gisela Terwindt**, The Netherlands	
14:25-14:40	NO: **Katharina Eikermann-Haerter,** USA	
14:40-14:50	Discussion, Rebuttals and Post-Debate Voting	
14:50-15:40	**Oral anti-CGRP drugs will displace injectable ones in the prevention of migraine**	
	Capsule: Four injectable monoclonal antibodies targeting the CGRP pathway and two oral anti CGRP agents, share good evidence for efficacy and safety in the prevention of migraine. Whether migraineurs will prefer to take an injectable agent monthly or quarterly or an oral agent, remains to be seen.	
14:50-15:00	Introduction and Pre-Debate Voting	
15:00-15:15	YES: **Sait Ashina**, USA	
15:15-15:30	NO: **Uwe Reuter**, Germany	
15:30-15:40	Discussion, Rebuttals and Post-Debate Voting	
15:40-16:30	**Psilocybin will be useful for treating refractory primary headache disorders**	
	Capsule: Despite traditional preventive medications, botulinum toxin injections, and newer CGRP blocking agents, successful treatment is elusive for many chronic headache patients. Psilocybin-based compounds may be potential solutions.	
15:40-15:50	Introduction and Pre-Debate Voting	
15:50-16:05	YES: **Cristina Tassorelli,** Italy	
16:05-16:20	NO: **Avi Ashkenazi**, Israel	
16:20-16:30	Discussion, Rebuttals and Post-Debate Voting	

16:30-18:10	**HEADACHE**	**HALL A**
Chair:	**Giorgio Lambru**, UK	
16:30-17:20	**All potential adverse effects to medication should be discussed prior to prescribing**	
	Capsule: Ethically, patients have the right to know the risks of treatment, but does this extend to less dangerous or rare potential adverse effects?	
16:30-16:40	Introduction and Pre-Debate Voting	
16:40-16:55	YES: **Lars Bendtsen**, Denmark	
16:55-17:10	NO: **Morris Levin**, USA	
17:10-17:20	Discussion, Rebuttals and Post-Debate Voting	
17:20-18:10	**The combination of Onabotulinium toxin and CGRP mAb is superior to either agent alone in the prevention of chronic migraine. Yes or No?**	
	Capsule: Real world evidence suggests that Botulinum toxin type A is very useful in combination with anti-CGRP mAbs in the prevention of chronic migraine	
17:20-17:30	Introduction and Pre-Debate Voting	
17:30-17:45	YES: **Rami Burstein**, USA	
17:45-18:00	NO: **Hayrunnisa Bolay-Belen**, Turkey	
18:00-18:10	Discussion, Rebuttals and Post-Debate Voting	
18:10-19:50	**HEADACHE**	**HALL A**
Chair:	**Morris Levin,** USA	
18:10-19:00	**Is the post Covid-19 headache a secondary post infections headache or worsening of preexisting primary headache disorder?**	
	Capsule: The reports on neurological findings in COVID-19 infection are increasing rapidly and headache seems to be the leader on the symptom list. Is persistent headache a new post-infectious entity?	
18:10-18:20	Introduction and Pre-Debate Voting	
18:20-18:35	Secondary: **Giorgio Lambru**, UK	
18:35-18:50	Worsening: **Dimos Mitsikostas**, Greece	
18:50-19:00	Discussion, Rebuttals and Post-Debate Voting	

19:00-19:50	**Do triptans demonstrate better efficacy and/or tolerability than ditans or gepants?**
	Capsule: New is not always better and data suggest that ditans and gepants are not more potent than triptans. But have meaningful outcomes and real evidence been considered?
19:00-19:10	Introduction and Pre-Debate Voting
19:10-19:25	YES: **Patricia Pozo-Rosich**, Spain
19:25-19:40	NO: **Bojana Zvan**, Slovenia
19:40-19:50	Discussion, Rebuttals and Post-Debate Voting

FRIDAY, MARCH 25, 2022		
14:00-15:40	**PARKINSON'S DISEASE (PD) I**	**HALL B**
Chair:	**Stuart H. Isaacson**, USA	
14:00-14:50	**Covid-19 is the perfect storm for emergence of parkinsonism**	
	Capsule: Evidence suggests that Covid-19 can aggravate specific motor and non-motor symptoms of PD. But could the mechanisms behind the infection play a role in the emergence of a novel parkinsonian syndrome?	
14:00-14:10	Introduction and Pre-Debate Voting	
14:10-14:25	YES: **K Ray Chaudhuri**, UK	
14:25-14:40	NO: **Angelo Antonini**, Italy	
14:40-14:50	Discussion, Rebuttals and Post-Debate Voting	
14:50-15:40	**Subjective cognitive decline is important to recognize in the cognitive spectrum of PD**	
	Capsule: Subjective cognitive decline is a self-perceived decline in cognitive ability reported in association with normal performance on daily activities and standardised cognitive tests. Its prognostic value in predicting objective cognitive decline has been suggested in the context of Alzheimer's disease. Could the same be true for PD-associated cognitive decline?	
14:50-15:00	Introduction and Pre-Debate Voting	
15:00-15:15	YES: **Lucia Batzu**, UK	
15:15-15:30	NO: **Eleonora Fiorenzato**, Italy	
15:30-15:40	Discussion, Rebuttals and Post-Debate Voting	

15:40-17:20	**PARKINSON'S DISEASE (PD) I**	**HALL B**
Chair:	**Angelo Antonini,** Italy	
15:40-16:30	**We need an algorithm to manage advanced PD**	
	Capsule: Advanced PD refers to the stage of disease when motor complications are difficult to manage with standard therapy. Patients reaching this stage of the disease may benefit from switching from oral to device-aided therapies. Is there a need to use specific clinical indicators to assess the eligibility for device-aided therapies and can the treatment be chosen on the basis of standardised and unanimous agreed criteria?	
15:40-15:50	Introduction and Pre-Debate Voting	
15:50-16:05	YES: **Stuart H. Isaacson**, USA	
16:05-16:20	NO: **Per Odin**, Sweden	
16:20-16:30	Discussion, Rebuttals and Post-Debate Voting	
16:30-17:20	**Objective monitoring in PD will be the norm in the future and is preferable to clinician-based assessment**	
	Capsule: Over the last decade, a growing number of researchers and clinicians have used advanced technologies, including wearable sensors, for objective monitoring of specific symptoms in patients with PD. Will objective monitoring become a worldwide standardised assessment tool for PD and will its use improve patients' management?	
16:30-16:40	Introduction and Pre-Debate Voting	
16:40-16:55	YES: **Anat Mirelman**, Israel	
16:55-17:10	NO: **Cristian Falup Pecurariu**, Romania	
17:10-17:20	Discussion, Rebuttals and Post-Debate Voting	
17:20-19:00	**PARKINSON'S DISEASE (PD) I**	**HALL B**
Chair:	**K. Ray Chaudhuri**, UK	
17:20-18:10	**Using probiotics is a waste of time in PD management.**	
	Capsule: It has been postulated that gut pathogens can contribute to the pathophysiological mechanisms behind PD and its clinical phenotype. However, does the use of probiotics to address motor and non-motor symptoms of PD represent a utopia and offer false hopes to patients?	
17:20-17:30	Introduction and Pre-Debate Voting	
17:30-17:45	YES: **Bogdan Ciopleias**, Romania	

17:45-18:00	NO: **Valentina Leta**, UK	
18:00-18:10	Discussion, Rebuttals and Post-Debate Voting	
18:10-19:00	**PARKINSON'S DISEASE (PD) I**	**HALL B**
	Genetics has not advanced care for PD patients	
	Capsule:The genetic architecture of PD is characterised by monogenic mutations causing familial disease, genetic variants increasing PD risk in specific populations and variants that contribute to increase the risk of developing sporadic PD. Are the clinical implications of this knowledge still insufficient to improve the clinical management of people with PD?	
18:10-18:20	Introduction and Pre-Debate Voting	
18:20-18:35	YES: **Nicola Pavese,** UK	
18:35-18:50	NO: **Heinz Reichmann**, Germany	
18:50-19:00	Discussion, Rebuttals and Post-Debate Voting	

FRIDAY, MARCH 25, 2022		
14:00-16:30	**MULTIPLE SCLEROSIS (MS)**	**HALL C**
Chair:	**Klaus Schmierer**, UK	
14:00-14:50	**Lifestyle interventions are as relevant for disease control as disease modifying therapies (DMTs) *in MS***	
	Capsule: There are currently close to twenty disease-modifying therapies for patients with MS that have been shown to reduce the number of clinical relapses and, in some instances, slow down the accumulation of neurological disability. It was also demonstrated that environment factors account for susceptibility to developing MS, and possibly modifying the disease course. Which intervention has a better risk-benefit ratio: DMT or lifestyle modifications?	
14:00-14:10	Introduction and Pre-Debate Voting	
14:10-14:25	YES: **Agne Straukiene**, UK	
14:25-14:40	NO: **Kate Petheram**, UK	
14:40-14:50	Discussion, Rebuttals and Post-Debate Voting	

	MULTIPLE SCLEROSIS (MS)	**HALL C**
14:50-15:40	**The microbiome is a therapeutic target in MS**	
	Capsule: Environmental factors are thought to trigger and perpetuate inflammation in patients with MS. Preclinical data have shown that the gastrointestinal microbiome environment may play a critical role in regulating immune responses in autoimmunity in the CNS. Fecal transfer has been successfully used in humans with inflammatory bowel disease, and it is no stretch of the imagination to think that similar interventions could become available for MS patients. Do preclinical data provide sufficient plausibility to pursue this route of inquiry? What is the magnitude of the effect of the microbiome in MS inflammation? What role do approved DMTs play in microbiome biology?	
14:50-15:00	Introduction and Pre-Debate Voting	
15:00-15:15	YES: **Cris Constantinescu**, UK	
15:15-15:30	NO: **Patrick Vermersch**, France	
15:30-15:40	Discussion, Rebuttals and Post-Debate Voting	
15:40-16:30	**Hematopoietic stem cell therapies (HSCT) should be offered as a first line DMT in selected MS patients**	
	Capsule: Autologous HSCT is considered the most potent immunomodulatory intervention available for patients with active MS. There are also perceived benefits to initiating therapy as early as possible. Can it be assumed that HSCT is an ideal first line intervention for MS patients? Or should concerns about potential diagnostic and safety issues delay its use?	
15:40-15:50	Introduction and Pre-Debate Voting	
15:50-16:05	YES: **Mark Freedman,** Canada	
16:05-16:20	NO: **Jaime Imitola,** USA	
16:20-16:30	Discussion, Rebuttals and Post-Debate Voting	

16:30-19:00	**MULTIPLE SCLEROSIS (MS)**	**HALL C**
Chair:	**Olaf Stuve**, USA	
16:30-17:20	**Slowly expanding lesions (SELs) are clinically meaningful for disease progression**	
	Capsule: SELs in MS are a relatively recent discovery using both susceptibility-weighted MRI and neuropathology highlighting iron rims. SELs are part and parcel of the discussion around "smoldering MS", which is thought to underlie insidious disease progression without new inflammatory activity detectable using standard MRI. However, only a small proportion of lesions on T2 weighted MRI appear to represent SELs. Are they clinically meaningful?	
16:30-16:40	Introduction and Pre-Debate Voting	
16:40-16:55	YES: **Daniel Reich**, USA	
16:55-17:10	NO: **Maria Rocca**, Italy	
17:10-17:20	Discussion, Rebuttals and Post-Debate Voting	
17:20-18:10	**DMTs approved for progressive MS are only effective in active progressive MS**	
	Capsule: Ocrelizumab and Siponimod are licensed not only for relapsing MS, but also for progressive disease. However, disease activity needs to be demonstrated on MRI to underpin their usefulness in people with progressive MS. Is such restriction justified, or does the latest evidence also support their use in people with MS with no demonstrable inflammatory changes on MRI?	
17:20-17:30	Introduction and Pre-Debate Voting	
17:30-17:45	YES: **Gilles Edan**, France	
17:45-18:00	NO: **Xavier Montalban,** Spain	
18:00-18:10	Discussion, Rebuttals and Post-Debate Voting	
18:10-19:00	**To withhold effective MS treatment cannot be ethically justified**	
	Capsule: Watching and waiting used to be a common approach in the (non-) treatment of people with MS. However, the advent of effective disease-modifying treatments challenge this strategy. On what grounds – if any – is it nowadays justified to withhold DMT from a person with MS provided there are no contraindications?	
18:10-18:20	Introduction and Pre-Debate Voting	
18:20-18:35	YES: **Leonora Fisniku,** UK	
18:35-18:50	NO: **Olaf Stuve**, USA	
18:50-19:00	Discussion, Rebuttals and Post-Debate Voting	

19:00-19:50	**European Charcot Foundation**
Chairs:	**Giancarlo Comi,** Italy **Per Soelberg Soerensen,** Denmark
	Assessing treatment response in progressive MS
	Capsule: Progressive MS is still an incurable disease but several interventions have been suggested. Since the disease is advancing very slowly, biomarkers have been suggested to replace clinical evidence of benefit, including imaging, visual parameters, plasma or cerebrospinal fluid protein levels.
19:00-19:01	Introduction
19:01-19:16	Role of MRI **Nicola De Stefano,** Italy
19:16-19:31	Role of visual platform **Letizia Leocani**, Italy
19:31-19:46	Role of body fluid biomarkers **Hans-Peter Hartung**, Germany
19:46-19:50	Final discussion

FRIDAY, MARCH 25, 2022		
14:00-15:40	**STROKE**	**HALL D**
Chair:	**Jesse Dawson**, UK	
14:00-14:50	**All people with ischemic stroke should receive long-term monitoring (at least 30 days) to detect paroxysmal atrial fibrillation (PAF).**	
	Capsule: Long term monitoring for PAF is known to increase detection rate in people with cryptogenic ischemic stroke. However rates of AF detection are typically higher in older people and several studies demonstrate similar rates of detection regardless of stroke etiology. In addition, whether long term monitoring detects clinically meaningful AF is unclear and it has not yet been known whether this improves outcomes and prevent recurrent strokes.	
14:00-14:10	Introduction and Pre-Debate Voting	
14:10-14:25	Yes: **Ante Anic,** Croatia	
14:25-14:40	No: **Zoltan Csanadi**, Hungary	
14:40-14:50	Discussion, Rebuttals and Post-Debate Voting	

14:50-15:40	**Is brain stimulation more efficient than pharmacological modulation for improving neurorecovery after stroke?**	
	Capsule: Neuromodulation techniques are being developed to improve a variety of outcomes following acute stroke and to improve post-stroke recovery. For example, the use of a paired vagus nerve stimulation system has recently been approved by the FDA to treat people with long-term arm impairment after ischemic stroke. In the last years, there has been an accumulated body of evidence that pharmacological modulation improved consistently clinical neurorecovery after stroke. Which approach is most likely to be successful?	
14:50-15:00	Introduction and Pre-Debate Voting	
15:00-15:15	YES: **Jesse Dawson**, UK	
15:15-15:30	NO: **Dafin Muresanu**, Romania	
15:30-15:40	Discussion, Rebuttals and Post-Debate Voting	
15:40-18:10	**STROKE**	**HALL D**
Chair:	**Laszlo Csiba,** Hungary	
15:40-16:30	**Can we mitigate the risk of stroke due to tobacco consumption by other methods of tobacco use without combustion?**	
	Capsule: Smoking tobacco is an established risk factor for stroke as well as for other cardiovascular and non-cardiovascular diseases. The best way to prevent smoking related disease (and stroke) is to avoid smoking. However it is very difficult for actual smokers to kick off the habit. This panel is intended to review and debate whether other methods of tobacco use (without combustion) could affect smoking related disease and whether those methods should be accepted by regulatory authorities.	
15:40-15:50	Introduction and Pre-Debate Voting	
15:50-16:05	Yes: **Hovhannes Manvelyan**, Armenia	
16:05-16:20	No**: Dov Gavish**, Israel	
16:20-16:30	Discussion, Rebuttals and Post-Debate Voting	
16:30-17:20	**Minimally invasive surgery should be preferred to conventional neurosurgery for hematoma evacuation after intracerebral hemorrhage (ICH)**	
	Capsule: Minimally invasive surgery for ICH is a promising treatment and is routinely performed in many centers. However, the largest clinical trials have not demonstrated definitive benefit of minimal surgical intervention on mortality and functional outcome	
16:30-16:40	Introduction and Pre-Debate Voting	
16:40-16:55	YES**: Adrian Parry Jones**, UK	
16:55-17:10	NO: **Wendy Ziai**, USA	
17:10-17:20	Discussion, Rebuttals and Post-Debate Voting	

17:20-18:10	**Tenecteplase should be routinely used for thrombolysis in acute ischemic stroke**	
	Capsule: Tenecteplase has a longer half-life than alteplase and can be given as a single bolus, but no trial has demonstrated superiority over alteplase and the largest trial to date included mostly people with minor stroke. Other studies suggest better recanalization rates with Tenecteplase, perhaps due to greater fibrin specificity. However, in most countries this remains an off-label treatment.	
17:20-17:30	Introduction and Pre-Debate Voting	
17:30-17:45	YES: **Melinda Roaldsen**, Norway	
17:45-18:00	NO: **Ashfaq Shuaib,** Canada	
18:00-18:10	Discussion, Rebuttals and Post-Debate Voting	
18:10-19:50	**STROKE**	**HALL D**
Chair:	**Ashfaq Shuaib,** Canada	
18:10-19:00	**In the era of carotid artery angiography (CTA) and magnetic resonance angiography (MRA), carotid ultrasound still has a role in decision making in people with carotid artery stenosis**	
	Capsule: It remains to be determined whether the impact of plaque characteristics on procedural risks differs between carotid artery stenting (CAS) and endarterectomy (CEA). In spite of some prospective multicentre studies (e.g. ICAROS) it is still controversial whether assessment of carotid plaque echolucency, ulceration, gray-scale-median, contrast-enhancement, jelly fish sign etc. predict the risk of embolism during CAS or CEA	
18:10-18:20	Introduction and Pre-Debate Voting	
18:20-18:35	Yes: **Laszlo Csiba**, Hungary	
18:35-18:50	No: **Adnan Siddiqui**, USA	
18:50-19:00	Discussion, Rebuttals and Post-Debate Voting	

19:00-19:50	**Withdrawal of care should never be performed within the first few days after severe acute stroke**	
	Capsule: *Discussions around withdrawal of care are common in the early days after severe stroke. Many patients have previously expressed the view that they would not wish to live with severe disability after stroke. However, outcomes at this early stage can be hard to predict and the intensity of medical treatment is related to risk of death. Therefore there are concerns that withdrawal of care will lead to a self fulfilling prophecy of increased risk of death.*	
19:00-19:10	Introduction and Pre-Debate Voting	
19:10-19:25	YES: **Gillian Mead**,UK	
19:25-19:40	NO: **Ruth England**, UK	
19:40-19:50	Discussion, Rebuttals and Post-Debate Voting	
FRIDAY, MARCH 25, 2022		
14:00-17:20	**ALZHEIMER'S DISEASE (AD) AND DEMENTIA**	**HALL E**
Chair:	**Robert Perneczky**, Germany	
14:00-14:50	**Is intracellular tau a better treatment target than extracellular tau**	
	Capsule: The microtubule-associated, axonal tau protein accumulates both in the intra- and the extracellular space in AD and other tauopathies. Current disease-modifying immunotherapies mostly target the extracellular protein to inhibit the spreading of tau pathology in the brain. So far, all clinical studies focusing on extracellular tau have failed. The present debate will focus on the pros and cons of extra- vs intracellular tau as a promising treatment target.	
14:00-14:10	Introduction and Pre-Debate Voting	
14:10-14:25	YES: **Einar Sigurdsson**, USA	
14:25-14:40	NO: **Ravi Jagasia**, Switzerland	
14:40-14:50	Discussion, Rebuttals and Post-Debate Voting	

	ALZHEIMER'S DISEASE (AD) AND DEMENTIA	**HALL E**
14:50-15:40	**Viruses are a major cause of amyloid deposition in the brain**	
	Capsule: Strong evidence exists for an association between herpes simplex virus type 1 (HSV1) and AD. Very recent epidemiological studies indicate that antiviral treatment for serious HSV1 infections confers protection against AD/dementia, supporting a causal role for the virus.	
14:50-15:00	Introduction and Pre-Debate Voting	
15:00-15:15	YES: **Ruth Itzhaki**, UK	
15:15-15:30	NO: **Magda Tsolaki**, Greece	
15:30-15:40	Discussion, Rebuttals and Post-Debate Voting	
15:40-16:30	**Should findings from monogenic dementias inform sporadic disease research?**	
	Capsule: After age, genes are the second-greatest factors associated with risk of AD. Children of parents with dementia have an increased lifetime risk of symptomatic AD, with greatest risk in those with a maternal history or with two affected parents, in particular if they inherited copies of the APOE ε4 allele. However, in contrast to familial AD, the sporadic form is not associated with mutations in the PSEN1, PSEN2 or APP genes and there is a debate on the usefulness of evidence from monogenic disease to inform sporadic AD research, to be discussed here.	
15:40-15:50	Introduction and Pre-Debate Voting	
15:50-16:05	YES**: Johannes Levin**, Germany	
16:05-16:20	NO: **Amos D. Korczyn**, Israel	
16:20-16:30	Discussion, Rebuttals and Post-Debate Voting	
16:30-17:20	**Is it too early to praise the benefits of gene editing for treatment of sporadic AD?**	
	Capsule: Familial AD is linked with mutations on the PSEN1, PSEN2 and APP genes, resulting in overexpression of pathological amyloid protein. The CRISPR/Cas9 genome editing tool is a powerful technology for correcting inconsistent genetic signatures, offering promising new treatment options for mutation carriers. However, sporadic AD is caused by impaired amyloid clearance from the brain without relevant overproduction. Therefore, it is questionable if gene editing is also promising in sporadic cases, which will be discussed in the present debate.	
16:30-16:40	Introduction	
16:40-17:10	**George Perry**, USA	
17:10-17:20	Discussion	

17:20-19:00	**ALZHEIMER'S DISEASE (AD) AND DEMENTIA**	**HALL E**
Chair	**Jonathan Kennedy**, UK	
17:20-18:10	**Should a person living with advanced stage dementia be allowed to request medical aid in dying while they are still competent, but have it triggered when cognitive function worsens?**	
	Capsule: A person develops advanced stage dementia. Years prior, before (s)he had dementia, she expresses in writing that (s)he does not want to live with this stage dementia. Is it ethical to allow advance care directives that instruct physicians to hasten own death when cognitive problems worsen?	
17:20-17:30	Introduction and Pre-Debate Voting	
17:30-17:45	YES: **Andrew Peterson**, USA	
17:45-18:00	NO: **Marie Nicolini**, USA	
18:00-18:10	Discussion, Rebuttals and Post-Debate Voting	
18:10-19:00	**Does the evidence support a role for the gut microbiome in dementia pathogenesis?**	
	Capsule: The gut microbiota comprises a complex community of microorganism species residing in the human gastrointestinal system. Changes of the microbiome may not only be associated with various gut disorders but also with brain diseases such as Parkinson's disease and AD. The increased permeability of the gut and blood-brain barrier induced by microbiota dysbiosis may mediate or affect AD pathogenesis, and imbalances in the gut microbiota could induce neuroinflammation. The present debate will focus on the question if there is enough evidence available to suggest a significant role of the gut microbiome for AD development.	
18:10-18:20	Introduction and Pre-Debate Voting	
18:20-18:35	YES: **Nikhil Sharma**, UK	
18:35-18:50	NO: **Julian Griffin,** UK	
18:50-19:00	Discussion, Rebuttals and Post-Debate Voting	

19:00-19:50	ALZHEIMER'S DISEASE (AD) AND DEMENTIA	HALL E
19:00-19:50	**Do vascular lesions contribute directly to AD pathology?**	
	Capsule: Epidemiological, clinical, and pathological evidence suggest a considerable overlap between cerebrovascular lesions and AD. Both pathologies could have additive and/or synergistic effects on cognitive decline and dementia. Cerebral amyloid angiopathy and small vessel disease are the most frequent vascular pathologies in older age and AD. However, there is an ongoing debate if vascular lesions contribute directly to amyloid pathology, which will be discussed in the present debate.	
19:00-19:10	Introduction and Pre-Debate Voting	
19:10-19:25	YES: **Ophir Keret**, Israel	
19:25-19:40	NO: **Raj Kalaria** UK	
19:40-19:50	Discussion, Rebuttals and Post-Debate Voting	

SATURDAY, MARCH 26, 2022		
14:00-15:40	**NEUROIMMUNOLOGY**	**HALL A**
Chair:	**Abhijit Chaudhuri,** UK	
14:00-14:50	**Are placebo-controlled trials still ethical for neuromyelitis optica spectrum disorders (NMOSD) in the current therapeutic environments?**	
	Capsule: There are now approved and highly effective treatments for NMOSD. Given their high efficacy, head to head trials to establish superiority are not feasible. Does the current therapeutic milieu preclude placebo-controlled studies of new agents even if the drug being tested has lesser toxicity or cost?	
14:00-14:10	Introduction and Pre-Debate Voting	
14:10-14:25	YES: **Hans-Peter Hartung,** Germany	
14:25-14:40	NO: **Alicja Kalinowska**, Poland	
14:40-14:50	Discussion, Rebuttals and Post-Debate Voting	

14:50-15:40	**Routine MRI surveillance should be undertaken in patients with confirmed or suspected NMOSD and MOGAD to assess disease activity**	
	Capsule: Routine MRI surveillance is routinely performed in patients with MS, and is a valuable outcome for phase 2 clinical trials. Brain and spinal cord lesions may develop in the absence of clinical symptoms in NMOSD and MOGAD, but it is believed that the frequency is low. Is there value of routine MRI surveillance of either the brain or spinal cord in these conditions?	
14:50-15:00	Introduction and Pre-Debate Voting	
15:00-15:15	YES: **Friedemann Paul**, Germany	
15:15-15:30	NO: **Saif Huda,** UK	
15:30-15:40	Discussion, Rebuttals and Post-Debate Voting	
15:40-17:20	**NEUROIMMUNOLOGY**	**HALL A**
Chair:	**Friedemann Paul**, Germany	
15:40-16:30	**All patients with autoimmune encephalitis should undergo comprehensive examination and, if negative, repeated testing for an underlying neoplasm regardless of the nature of the associated autoantibody**	
	Capsule: Often patients with autoimmune encephalitis have underlying neoplasms; identification and treatment of the cancer may have therapeutic benefit. However, in many patients, search for occult neoplasms is negative and frustrating. Should a search for an underlying neoplasm be routinely instituted in all patients with a suspected diagnosis of autoimmune encephalitis?	
15:40-15:50	Introduction and Pre-Debate Voting	
15:50-16:05	YES: **Amy Kunchok,** USA	
16:05-16:20	NO: **Sarosh Irani,** UK	
16:20-16:30	Discussion, Rebuttals and Post-Debate Voting	
16:30-17:20	**All patients with new onset psychosis should be investigated for and, unless other cause is found, should be treated empirically with corticosteroids for potential autoimmune encephalitis**	
	Capsule: Some patients with new onset psychosis with no premorbid features of a psychiatric illness may be suspected of having an autoimmune encephalitis. Occasionally, MRI may be normal in patients with autoimmune encephalitis. Should patients with such clinical features and no obvious underlying psychiatric disorder or infectious encephalitis be treated empirically for possible autoimmune encephalitis?	
16:30-16:40	Introduction and Pre-Debate Voting	
16:40-16:55	YES: **Abhijit Chaudhuri,** UK	
16:55-17:10	NO: **Anastasia Zekeridou,** USA/Greece	
17:10-17:20	Discussion, Rebuttals and Post-Debate Voting	

17:20-19:50	**NEUROIMMUNOLOGY**	**HALL A**
Chair:	**Brian Weinshenker**, USA	
17:20-18:10	**Checkpoint-inhibitor-associated autoimmune conditions should be routinely managed by discontinuation of the checkpoint inhibitor.**	
	Capsule: Checkpoint inhibitors may result in autoimmune conditions, including CNS inflammatory disorders. However, discontinuation of treatment may result in progression of the cancer for which the patient is receiving the drug. Should checkpoint inhibitors be routinely discontinued when patients develop an autoimmune condition after treatment?	
17:20-17:30	Introduction and Pre-Debate Voting	
17:30-17:45	YES: **Anastasia Zekeridou**, USA/Greece	
17:45-18:00	NO: **Uros Rot,** Slovenia	
18:00-18:10	Discussion, Rebuttals and Post-Debate Voting	
18:10-19:00	**Persistent seropositivity of MOG-IgG antibody requires immunosuppressive therapy even in the absence of clinical relapse**	
	Capsule: In some patients with MOG-IgG-associated diseases, relapse may not occur. It is generally agreed that relapse is an indication for institution of maintenance treatment. But it has also been suggested that persistent seropositivity for MOG-IgG may also predict relapse. Should persistent seropositivity be an indication for institution of maintenance immunosuppressive therapy in the absence of attack?	
18:10-18:20	Introduction and Pre-Debate Voting	
18:20-18:35	YES: **Michael Levy**, USA	
18:35-18:50	NO: **Eoin P. Flanagan**, USA	
18:50-19:00	Discussion, Rebuttals and Post-Debate Voting	

19:00-19:50	**All anti-CD19 and anti-CD20 drugs have similar efficacy. None offers significant advantages compared to others in the same class.**	
	Capsule: B-cell depletion is highly effective for MS and AQP4-IgG seropositive NMOSD, as well as for other autoimmune conditions including those affecting the CNS. Recently, inebilizumab, a CD-19 targeting mAb has been approved for AQP4-IgG seropositive NMOSD. Is there reason to believe that the specific B-cell target (CD19 or CD20) or other differences between anti-CD20 drugs (e.g. humanized vs chimeric) influence efficacy and justify the higher cost of newer agents?	
19:00-19:10	Introduction and Pre-Debate Voting	
19:10-19:25	YES: **David Baker**, UK	
19:25-19:40	NO: **Brian Weinshenker**, USA	
19:40-19:50	Discussion, Rebuttals and Post-Debate Voting	
19:50-20:00	**CLOSING SESSION**	**HALL A**
19:50-19:55	Closing remarks: **Amos D. Korczyn**, Israel	
19:55-20:00	Invitation to CONy 2023: **Vida Demarin**, Croatia	

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SATURDAY, MARCH 26, 2022		
14:00-15:40	**EPILEPSY**	**HALL B**
Chair:	**Alla Guekht**, Russia	
14:00-14:50	**When a medication has failed to control seizures, should a medication with a different mechanism of action be preferentially prescribed as the next choice?**	
	Capsule: Several mechanisms of action have been elucidated for antiseizure medication. Some share similar mechanisms, e.g., sodium channel blockers, GABA receptor agonists, SV2A blockers. When one drug with a known mechanism of action has failed to control seizures, should a drug with a different mechanism be preferentially used next in the sequence of treatment?	
14:00-14:10	Introduction and Pre-Debate Voting	
14:10-14:25	Yes: **Emilio Perucca**, Australia	
14:25-14:40	No: **Matthew Walker**, UK	
14:40-14:50	Discussion, Rebuttals and Post-Debate Voting	
14:50-15:40	**Do seizure detection devices have a significant role in managing people with epilepsy?**	
	Capsule: Several devices now exist that can detect seizures through non-invasive means, including those that can be worn on the wrist like a smart watch. They have reasonably high sensitivity and specificity. Should they be broadly used in managing the care of people with epilepsy?	
14:50-15:00	Introduction and Pre-Debate Voting	
15:00-15:15	Yes: **Sandor Beniczky**, Denmark	
15:15-15:30	No: **Andreas Schulze-Bonhage,** Germany	
15:30-15:40	Discussion, Rebuttals and Post-Debate Voting	

15:40-17:20	**EPILEPSY**	**HALL B**
Chair:	**Matthew Walker**, UK	
15:40-16:30	**Are outcomes of randomized controlled trials (RCT's) relevant to treat patients or are "real world" trials more valuable?**	
	Capsule: Drugs are approved by governmental agencies only after undergoing rigorous experimental testing, typically in people with drug-resistant epilepsy. These trials have selective criteria for subject enrollment and results are carefully assessed. Are these trials adequate or are the methods so different from ordinary clinical practice that the results are not relevant for most patients treated in a typical outpatient setting?	
15:40-15:50	Introduction and Pre-Debate Voting	
15:50-16:05	RCT's: **Jacqueline French,** USA	
16:05-16:20	Real World: **Martin Brodie,** UK	
16:20-16:30	Discussion, Rebuttals and Post-Debate Voting	
16:30-17:20	**Should enzyme inducing antiseizure medications be avoided because of cardiovascular and other risks?**	
	Capsule: Enzyme inducing drugs have been shown to have unfavorable effects on serum lipids, C-reactive protein, and possibly cardiovascular risk. However, these agents are the least expensive drugs and often well-tolerated. Because of the potential for cardiovascular risk, should their use be abandoned?	
16:30-16:40	Introduction and Pre-Debate Voting	
16:40-16:55	Yes: **Ley Sander,** UK	
16:55-17:10	No: **Alla Guekht,** Russia	
17:10-17:20	Discussion, Rebuttals and Post-Debate Voting	

17:20-19:50	**EPILEPSY**	**HALL B**
Chair:	**Michael Sperling**, USA	
17:20-18:10	**Is it ethical to provoke psychogenic seizures for purposes of diagnosis?**	
	Capsule: Inpatient video-EEG monitoring is expensive, time-consuming, and often fails to record seizures during elective admissions. As psychogenic seizures can be provoked by suggestion techniques, this permits a more efficient means of rapidly obtaining a diagnosis in many patients. Whether this involves deception is a matter of debate. Is it ethical to use a suggestion technique?	
17:20-17:30	Introduction and Pre-Debate Voting	
17:30-17:45	Yes: **Curt LaFrance**, USA	
17:45-18:00	No: **Barbara Dworetzky**, USA	
18:00-18:10	Discussion, Rebuttals and Post-Debate Voting	
18:10-19:00	**Has genetic testing improved care for people with epilepsy?**	
	Capsule: Sophisticated genetic testing has become available at affordable prices in recent years. These provide specific information about the underlying genetic abnormality and should lead to precision in therapy. Has genetic testing lived up to its promise and does it provide clinically useful and meaningful information?	
18:10-18:20	Introduction and Pre-Debate Voting	
18:20-18:35	Yes: **Alica Goldman**, USA	
18:35-18:50	No: **Nicola Specchio**, Italy	
18:50-19:00	Discussion, Rebuttals and Post-Debate Voting	
19:00-19:50	**Should patients usually have a benzodiazepine rescue therapy plan in place to treat seizure clusters or prolonged seizures?**	
	Capsule*: Many individuals experience seizures in clusters of multiple seizures occurring within a day or two. Others have seizures that sometimes are prolonged. Benzodiazepine rescue therapy has been advocated and approved by regulatory authorities to treat such episodes. However, many seizures within clusters recur at prolonged intervals and perhaps other therapies might be equally effective without causing the sedative effects of benzodiazepines. Also, current methods of administration are associated with relatively slow absorption of these drugs (peaking in an hour or more). Should this one approach, using benzodiazepines, be generally advised for all who cluster or have prolonged seizures?*	
19:00-19:10	Introduction and Pre-Debate Voting	
19:10-19:25	Yes: **Manjari Tripathi,** India	
19:25-19:40	No: **Zeljka Petelin Gadze,** Croatia	
19:40-19:50	Discussion, Rebuttals and Post-Debate Voting	

SATURDAY, MARCH 26, 2022		
14:00-15:40	**AMYOTROPHIC LATERAL SCLEROSIS (ALS)**	**HALL C**
Chair:	**Nigel Leigh**, UK	
14:00-14:50	**Plasma neurofilament light (NfL) levels are now an essential addition to the ALS clinic**	
	Capsule: Measurements of plasma NfL levels are now widely available and may provide biomarkers for disease activity, progression and prognosis in ALS. However, their place in the ALS clinic is uncertain: Are they useful in diagnosis? If so, in what context? Do NfL levels have useful prognostic value? Can they help in monitoring response to therapy? Our debate will crystalise the evidence for and against the use of plasma measurements of NfL proteins in the ALS/neuromuscular disorders clinic.	
14:00-14:10	Introduction and Pre-Debate Voting	
14:10-14:25	Yes: **Henrik Zetterberg**, Sweden	
14:25-14:40	No: **Martin Turner**, UK	
14:40-14:50	Discussion, Rebuttals and Post-Debate Voting	
14:50-15:40	**The human is the only valid model for ALS drug discovery**	
	Capsule: Remarkable progress on the science of ALS pathophysiology has been made in the last decade. Yet similar progress has not been seen in the identification of successful ALS therapeutics. Since the mid 1990's much preclinical work depended on animal models of ALS with vanishingly few successful outcomes. More recently the emergence of various human models along with sophisticated approaches to human postmortem tissues/biofluids has surfaced. The discussants will provide arguments for and against these widely different approaches to ALS modeling and therapy identification.	
14:50-15:00	Introduction and Pre-Debate Voting	
15:00-15:15	Yes: **Albert Ludolph**, Germany	
15:15-15:30	No: **Lucie Bruijn**, UK	
15:30-15:40	Discussion, Rebuttals and Post-Debate Voting	

15:40-17:20	**AMYOTROPHIC LATERAL SCLEROSIS (ALS)**	**HALL C**
Chair:	**Jeffrey D. Rothstein,** USA	
15:40-16:30	**Should high calorie high fat diet be started at diagnosis in ALS?**	
	Capsule: Catabolism is a negative prognostic factor for ALS. There is amounting evidence that counteracting catabolism in ALS is beneficial for survival of ALS patients and motor function. The focus is on the time of intervention which includes definition of the treatment target, and the type of intervention.	
15:40-15:50	Introduction	
15:50-16:20	**Albert Ludolph**, Germany	
16:20-16:30	Discussion	
16:30-17:20	**Are the new treatments for spinal muscular atrophy (SMA), so successful in the young, also indicated for adults?** *Capsule: The recent identification of gene therapy for SMA has been one of the most profound therapy discoveries for neurology- to effectively cure a fatal infant disease of motor neurons. It is also one of the most expensive therapies in the whole of medical practice. SMA also affects adults as a slowly progressive yet disabling disorder. Should these remarkable therapies also be applied to adult onset SMA individuals?*	
16:30-16:40	Introduction and Pre-Debate Voting	
16:40-16:55	YES: **Susanne Petri**, Germany	
16:55-17:10	NOT ALWAYS: **Amos D. Korczyn**, Israel	
17:10-17:20	Discussion, Rebuttals and Pre-Debate Voting	

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SATURDAY, MARCH 26, 2022		
17:20-18:10	**NEURODEGENERATIVE DISEASES**	**HALL C**
Chair:	**Peter Jenner,** UK	
17:20-18:10	**Is chronic traumatic encephalopathy (CTE) a useful clinical entity?**	
	Capsule: *The term chronic traumatic encephalopathy (CTE), coined more than half a century ago, refers to exposure to traumatic brain injury related to sports or other sources, that subsequently leads to neurodegeneration. CTE nowadays may be overused to explain the onset of neurodegenerative disease in later life. Is it a useful clinical entity in medical practice?*	
17:20-17:30	Introduction and Pre-Debate Voting	
17:30-17:45	YES: **Dan Perl**, USA	
17:45-18:00	NO: **Amos D. Korczyn**, Israel	
18:00-18:10	Discussion, Rebuttals and Post-Debate Voting	
18:10-19:50	**NEURODEGENERATIVE DISEASES**	**HALL C**
Chair:	**Bogdan Popescu,** Romania	
18:10-19:00	**Given a diagnosis indicative of pre-clinical neurodegenerative disease imposes personal responsibility on the patient.**	
	Capsule: If someone receives a diagnosis of pre-clinical neurodegenerative disease (biomarker diagnosis without signs and symptoms of clinical impairment), is that person ethically obligated to share that information with others, in particular, close family and health care providers?	
18:10-18:20	Introduction and Pre-Debate Voting	
18:20-18:35	YES: **Josh Grill**, USA	
18:35-18:50	No: **Emily Largent**, USA	
18:50-19:00	Discussion, Rebuttals and Post-Debate Voting	
19:00-19:50	**Can fetal cell transplantation replace dying neurons in neurodegenerative disease?**	
	Capsule: Can the implantation of fetal cells in neurodegenerative disease take on the physiological role of endogenous neurons, restore function and survive in an environment that led to the death of the original cells?	
19:00-19:10	Introduction and Pre-Debate Voting	
19:10-19:25	YES: **Serge Przedborski,** USA	
19:25-19:40	NO: **Clive Svendsen,** USA	
19:40-19:50	Discussion, Rebuttals and Post-Debate Voting	

SATURDAY, MARCH 26, 2022		
14:00-15:30	**PARKINSON'S DISEASE (PD) II** *Session supported by unrestricted grants from Acadia, Amneal, Avion, Kyowa Kirin, Merz Therapeutics, Supernus*	**HALL D**
Chair:	**Stuart H. Isaacson**, USA	
14:00-14:45	**Apomorphine should replace levodopa as the gold standard therapy for PD**	
	Capsule: Apomorphine is a dopamine agonist that has been used to treat PD symptoms for as long as levodopa. Despite poor bioavailability of oral levodopa and variable absorption, levodopa has been considered the 'gold standard' therapy. Apomorphine has similar robust efficacy and may provide more reliable benefit. Should our gold standard be changed to apomorphine?	
14:00-14:05	Introduction and Pre-Debate Voting	
14:05-14:15	YES: **Richard Dewey Jr**, USA	
14:15-14:25	NO: **Fernando Pagan**, USA	
14:25-14:45	Discussion, Rebuttals and Post-Debate Voting	
14:45-15:30	**Delayed ON is more important contributor to daily OFF time than end-dose wearing off**	
	Capsule: Despite advances to improve both peripheral levodopa pharmacokinetics and striatal dopaminergic activity, OFF time can persist. Traditionally, clinical focus has been on end-dose wearing off benefit. Recognition of symptom benefit onset, magnitude, duration, and reliability due to variable gastrointestinal oral levodopa delivery and transport is often overlooked. Can shifting clinical focus to identifying delayed onset of oral levodopa doses help improve OFF time management?	
14:45-14:50	Introduction and Pre-Debate Voting	
14:50-15:00	YES: **Daniel Kremens**, USA	
15:00-15:10	NO: **Yasar Torres-Yaghi**, USA	
15:10-15:30	Discussion, Rebuttals and Post-Debate Voting	

15:30-16:15	**Expert Roundtable: Clinical approach to Parkinson's disease (PD) psychosis** *Supported by an unrestricted educational grant from Acadia*
	Capsule: PD psychosis (PDP) is common, but difficult to treat and disproportionately impacts daily activities and quality of life. Yet traditional views have characterized PDP as 'benign' and treatment is often initiated only after significant morbidity occurs. Is it time to reconsider our PDP management paradigm?
Moderator:	**Stuart H. Isaacson**, USA
15:30-15:40	Screening and diagnosis: **Danielle Larson**, USA
15:40-15:50	PDP treatment paradigm: **Richard Dewey Jr**, USA
15:50-16:00	Impact of PDP and decision to treat: **Rajesh Pahwa**, USA
16:00-16:15	Live Discussion (faculty)
16:15-17:00	**Meet the Expert Roundtable** *Supported by an unrestricted educational grant from Avion*
	Meet the Expert Roundtable: Fine tuning PD medication regimens with fractional carbidopa/levodopa tablets
	Capsule: Motor complications are common, with many patients experiencing both peak dose dyskinesia and off episodes. Adjusting baseline levodopa dosing can sometimes be complicated using traditional 25mg/100mg half or whole tablets of IR CD/LD. Would finer tuning with quarter tablet dosing increments (6.25mg/25mg) improve symptom management with CD/LD?
Moderator:	**Stuart H. Isaacson**, USA
16:15-16:25	Overview of fractional carbidopa/levodopa tablets: **Rajesh Pahwa**, USA
16:25-16:35	Clinical scenarios with finer tuning to improve symptom control: **Yasar Torres-Yaghi**, USA
16:35-16:45	Early use experience with fractional carbidopa/levodopa: **Daniel Kremens**, USA
16:45-17:00	Live Discussion (faculty)

SATURDAY, MARCH 26, 2022		
17:00-19:15	**PARKINSON'S DISEASE (PD) II** *Session supported by unrestricted grants from Acadia, Amneal, Avion, Kyowa Kirin, Merz Therapeutics, Supernus*	**HALL D**
Chair:	**Stuart H. Isaacson,** USA	
17:00-17:45	**Sialorrhea evaluation and treatment should be prioritized at clinic visits**	
	Capsule: Sialorrhea is a common symptom of PD, yet its impact is often minimized during clinical visits. Chronic sialorrhea has been associated with physical consequences, psychosocial stigma, and potentially significant morbidity. Treatment with cholinergic denervation in salivary glands with botulinum toxin has become readily available first-line therapy. Should query and early treatment of sialorrhea be prioritized at routine clinic visits?	
17:00-17:05	Introduction and Pre-Debate Voting	
17:05-17:15	YES: **Fernando Pagan**, USA	
17:15-17:25	NO: **Fiona Gupta**, USA	
17:25-17:45	Discussion, Rebuttals and Post-Debate Voting	
17:45-18:30	**Treatment of motor fluctuations should begin with levodopa regimen changes before adding adjunctive polypharmacy**	
	Capsule: Levodopa is the cornerstone therapy for PD motor symptoms, yet motor fluctuations invariably emerge leading to many hours without motor benefit from levodopa doses (off episodes). Rational polypharmacy with adjunctive medications has been a common treatment strategy, yet may be limited by tolerability and persistent OFF time. Should newer extended levodopa formulations be used before adding adjunctive medications?	
17:45-17:50	Introduction and Pre-Debate Voting	
17:50-18:00	YES: **Richard Dewey Jr.**, USA	
18:00-18:10	NO: **Rajesh Pahwa**, USA	
18:10-18:30	Discussion, Rebuttals and Post-Debate Voting	

18:30-19:15	**Effective management of daily OFF time requires both dopaminergic and non-dopaminergic co-therapies**
	Capsule: Despite continuous levodopa delivery strategies, OFF time persists in most patients. This may indicate the limitations of presynaptic pathways to fully resolve OFF episodes. Striatal adenosine and glutamatergic receptors are overactive in PD, and impact direct and/or indirect pathway activity. Should nondopaminergic receptor antagonists be added to levodopa as soon as motor fluctuations emerge?
18:30-18:35	Introduction and Pre-Debate Voting
18:35-18:45	YES: **Danielle Larson**, USA
18:45-18:55	NO: **Yasar Torres-Yaghi**, USA
18:55-19:15	Discussion, Rebuttals and Post-Debate Voting
19:15-19:30	**Recap of Parkinson's Disease II and Closing Remarks** **Stuart H. Isaacson,** USA

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INDUSTRY SPONSORED PROGRAM

FRIDAY, MARCH 25, 2022		
13:00-13:50	**MEET THE EXPERT SESSION**	**MEET THE EXPERT**
	Practical use of apomorphine infusion – lessons from the clinic *Supported by Britannia Pharmaceuticals*	
13:00-13:05	Welcome & Introduction **K. Ray Chaudhuri**, UK	
13:05-13:35	Practical use of apomorphine infusion – lessons from the clinic **Marc Vérin**, France	
13:35-13:50	Questions and Answers Facilitated by **K. Ray Chaudhuri,** UK	

SATURDAY, MARCH 26, 2022		
13:00-14:00	**INDUSTRY-SPONSORED SYMPOSIUM**	**INDUSTRY**
	Getting the timing right – the case for earlier use of device-aided therapy in advanced Parkinson's disease (PD) *Supported by Britannia Pharmaceuticals*	
13:00-13:10	Welcome & Introduction **K. Ray Chaudhuri**, UK	
13:10-13:30	Should we be considering and using device-aided therapies earlier in advanced PD? **Tobias Warnecke**, Germany	
13:30-13:50	Are device-aided therapies underutilised in advanced PD and, if so, why? **Per Odin,** Sweden	
13:50-14:00	Questions and Answers Facilitated by **K. Ray Chaudhuri,** UK	

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Sanofi has provided an independent medical educational grant in support of the Multiple Sclerosis program.

Neurelis has provided an independent medical educational grant in support of the Epilepsy program.