Thursday April 04, 2019	**Hall-CAJAL**
09:00-11:00	**SESSION 1 | OPENING SESSION**
Chairpersons:	**Natan Bornstein,** Israel & **Hans Hamburger,** The Netherlands
09:00-09:30	Welcome remarks: **Amos Korczyn,** Israel & **Exuperio Diez Tejedor,** Spain
09:30-10:00	The role of fungi in the etiology of multiple sclerosis: **Julian Benito-Leon,** Spain
10:00-10:30	Immune checkpoint inhibitors and neurological disease: **Olaf Stuve,** USA
10:30-11:00	What´s next: Immunotherapy of MS after the anti-CD20s: **Antonio García Merino**, Spain
11:00-11:30	***Coffee Break***
11:30-13:00	**SESSION 2 | PLENARY LECTURES**
Chairpersons:	**Xiao Ping Wang**, China & **Fenny Yudiarto,** Indonesia
11:30-12:00	Is imagination a distinct metacognitive process with its own neurobiological substrate? **Daniel Drubach,** USA
12:00-12:30	The secrets of FXTAS: **Sharon Hassin-Baer,** Israel
12:30-13:00	The new world of focused ultrasound to treat neurodegenerative diseases: **Jose Obeso,** Spain
13:00-13:45	***Lunch Break***
13:45-15:15	**SESSION 3 | PLENARY LECTURES: EPILEPSY**
Chairpersons:	**Mar Carreno**, Spain & **Vladimir Donath,** Slovakia
13:45-14:15	Epilepsy genetics and precision therapies – trials and tribulations: **Samuel Berkovic,** Australia
14:15-14:45	Gene therapy in epilepsy: **Matthew Walker,** UK
14:45-15:15	How will new devices impact the diagnosis and treatment of seizures? **Michael Sperling,** USA
15:15-15:30	***Coffee Break***
15:30-18:00	**SESSION 4 | PLENARY LECTURES: ALZHEIMER'S DISEASE (AD)**
Chairpersons:	**Thashi Chang**, Sri Lanka **& George Perry**, USA
15:30-16:00	Beyond amyloid, the sweet trail to neuroprotection: **Stefano Sensi,** Italy
16:00-16:30	Tau seeding and disease progression in AD: **Isidro Ferrer**, Spain
16:30-17:00	Neuropathological basis of sleep disorders in neurodegenerative diseases: **Lea Grinberg,** USA/Brazil
17:00-18:00	**Debate: Is preclinical AD a useful term?** *Capsule: The diagnosis of AD has traditionally required both cognitive deterioration and certain pathological features, amyloid plaques and neurofibrillary tangles. However, the tissue changes appear decades before the clinical symptoms. Recently it has been suggested to term this stage as "preclinical AD". Is this a useful term?*
17:00-17:15	Host: **Giulio Maria Pasinetti,** USA
17:15-17:30	Yes: **David Knopman,** USA
17:30-17:45	No: **Amos Korczyn,** Israel
17:45-18:00	Discussions and rebuttals
18:00-19:00	**OPENING CEREMONY**

19:00	***Welcome Reception***

Friday April 05, 2019	**Hall - CAJAL**
07:30-08:30	**Meet the Experts–**(Lafora) **Integrating cladribine tablets into clinical practice** **Mark Freedman,** Canada **& Celia Oreja-Guevara,** Spain
07:30-08:30	**Meet the Expert-** (Lorente de Nó) **Targeting B cells in multiple sclerosis** **Ron Milo,** Israel
08:30-10:10	**SESSION 5 | MULTIPLE SCLEROSIS (MS): DIAGNOSIS**
Chairpersons:	**Mario Habek**, Croatia **& Manuel Seijo-Martinez,** Spain
08:30-09:20	**Will neurofilaments light (NFL) serum levels be the gold standard for monitoring MS progression, replacing MRI?** *Capsule: NFLs belong to the intermediate filament proteins family and are the major components of the cytoskeleton of neurons. Recent data suggest that NFL may be used as a prognostic factor to monitor disease progression, disease activity and treatment efficacy.*
08:30-08:40	Host: **Laszlo Vecsei**, Hungary
08:40-08:55	Yes: **Georgina Arrambide,** Spain
08:55-09:10	No:
09:10-09:20	Discussions and rebuttals
09:20-10:10	**Evoked potentials (EP's) still have a role in diagnosing MS and monitoring disease progression.** *Capsule: EP's have been used for a long time as diagnostic biomarkers for MS diagnosis but also recently considered beneficial for monitoring disease course and progression. Newer interventions on remyelination showed benefit of EP's on outcomes but did not support a clear improvement as measured with standard clinical outcomes. Should EP's be considered as surrogate measures for diagnosis and monitoring MS disease course?*
09:20-09:30	Host: **Jera Kruja,** Albania
09:30-09:45	Pro: **Letizia Leocani,** Italy
09:45-10:00	Con: **Bianca Weinstock-Guttman,** USA
10:00-10:10	Discussion and rebuttals
10:10-10:25	***Coffee Break***
10:25-12:05	**SESSION 6 | MS PATHOGENESIS**
Chairpersons:	**Oded Abramsky,** Israel, **& Fernando de Castro,** Spain
10:25-11:15	**Is immunosenescence a factor to be considered in treating patients older than 50?** *Capsule: Treatments for disease modification in MS are mostly studied in patient populations between 18 and 50. The immune system, the key target of our MS therapies, undergoes significant immune senescence. In addition, the influence of immune therapies on disease progression parameters show less influence of immune therapies on disability accrual, but potentially higher risks of these therapies with*

	aging.
10:25-10:35	Host:
10:35-10:50	Yes: **Mark Freedman**, Canada
10:50-11:05	No: **Joab Chapman**, Israel
11:05-11:15	Discussions and rebuttals
11:15-12:05	**Does primary progressive MS (PPMS) have the same immunopathogenesis as secondary progressive MS (SPMS)?** *Capsule: MS includes different clinical forms: relapsing remitting, secondary progressive or PPMS. The clinical manifestations of these forms of MS as well as the response to treatment vary substantially. Are the cause and immunopathogenesis the same or differ between MS patients subgroups?*
11:15-11:25	Host: **Ralf Linker,** Germany
11:25-11:40	Yes:
11:40-11:55	No: **Jacek Losy,** Poland
11:55-12:05	Discussions and rebuttals
12:15-13:15	**Industry Sponsored Symposium (Not for CME)-** **Transforming scientific innovation in MS into clinical practice** *Capsule: Data on the efficacy and safety of cladribine tablets in the treatment of RRMS will be presented, followed by an overview of the proposed MoA showing the selectivity of cladribine tablets in transiently reducing lymphocyte populations. Lastly, the speakers will debate how evolving treatments are helping in reducing MS disease burden.* **Mar Tintore,** Spain **-** Welcome and Introduction **Mar Tintore,** Spain**- cladribine tablets: translating innovative treatment approach into clinical practice** **Klaus Schmierer,** UK **- Does the selectivity of cladribine tablets explain the long-term outcomes?** **Celia Oreja-Guevara,** Spain **& Mar Tintore,** Spain **- Living without the burden of MS: fiction or reality?** **Mar Tintore,** Spain **- Q&A and meeting close**
13:15-14:15	***Lunch Break***
13:15-14:15	**Meet the Expert** (Rio Hortega) **AMBAR (Alzheimer´s Management By Albumin Replacement) Trial Results: Clinical and Biomarker Update** **Laura Núñez,** Spain **Javier Olazarán,** Spain
13:15-14:15	**Meet the Expert-** (Lorente de No) **Update on Clinical Use of Pimavanserin and PD Psychosis** **Rajesh Pahwa,** USA**; Fatta Nahab,** USA; **Daniel Kremens,**USA, **Stuart Isaacson,** USA
14:15-15:45	**SESSION 7 | MS THERAPY**
Chairpersons:	**Anas Jouhar,** Syria & **Mar Tintore,** Spain
14:15-14:55	**Should therapy be initiated in clinically isolated syndrome (CIS) cases not having oligoclonal bands (OCB)?** *Capsule: The existence of OCB in the CSF allows to predict a second clinical attack following a clinically*

	isolated syndrome (CIS) and now allows a diagnosis of MS, even without dissemination in time. Due to this, it becomes possible to prescribe early disease-modifying therapy (DMT) to patients with CIS. Is this justified?
14:15-14:25	Host: **Larysa Sokolova,** Ukraine
14:25-14:35	Yes: **Klaus Schmierer,** UK
14:35-14:45	No: **Marcin Mycko,** Poland
14:45:14:55	Discussions and rebuttals
14:55-15:45	**CSF is still important in the diagnosis of MS.- check If they can be earlier** *Capsule: The diagnosis of MS is based on demonstration of "lesions disseminated in time and space." Accordingly, diagnostic criteria have focused on clinical and MRI abnormalities that document "lesions." The CSF may establish the inflammatory and immunological nature of symptoms and help corroborate a diagnosis of MS. How specific and helpful are CSF findings? Do the costs, inconvenience and risks justify routine use of CSF to establish a diagnosis of MS?*
14:55-15:05	Host: **Uros Rot,** Slovenia
15:05-15:20	Pro: **Konrad Rejdak,** Poland
15:20-15:35	Con: **Brian Weinshenker**, USA
15:35-15:45	Discussions and rebuttals
15:45-16:00	Coffee Break

16:00-19:00	**SESSION 8 | ROLE OF CEREBROSPINAL FLUID (CSF)**
Chairpersons:	**Melchor Rodrigo,** Argentina & **Caroline Rush,** Canada
16:00-16:50	**Is the switch from brand-name to generic drugs in MS safe and justified?** *Capsule: As intellectual property protections are beginning to expire, cheaper generic drugs are entering the vibrant market. The complex structure of biologic drugs for MS or non-biologic complex drugs such as glatiramer acetate may make it difficult to reproduce them. Even minor changes in the manufacturing process may result in significant changes in the ultrastructure and biological properties of biosimilar. Are generics identical to, similar to or different from the original drugs?*
16:00-16:10	Host: **Ron Milo,** Israel
16:10-16:25	Yes: **Ovidiu Bajenaru,** Romania
16:25-16:40	No: **Klaus Schmierer,** UK
16:40-16:50	Discussion and rebuttals
16:50-17:40	**Cognitive dysfunction is amenable to MS specific disease modifying drugs (DMD).** *Capsule: Cognitive impairment (CI) occurs typically in neurodegenerative disease. Transient changes related to MS relapses are more recent observation. Strong evidence supports associations between MRI parameters and CI and therefore, worsening of defects on neuropsychological testing may also reflect disease activity. Should decline in cognition merit clinical attention when drugs are considered that may mitigate MS disease activity?*
16:50-17:00	Host: **Anastasios Orologas,** Greece
17:00-17:15	Pro: **Bianca Weinstock Guttman**, USA
17:15-17:30	Con: **Friedemann Paul,** Germany
17:30-17:40	Discussions and rebuttals

17:40-19:00	**Round table: The reasons of MS misdiagnosis**. Hosts : **Oscar Fernandez,** Spain **& Olaf Stuve**, USA Speakers: **Mark Freedman**, Canada**; Ralf Linker,** Germany**; Ron Milo**, Israel; **Bianca Weinstock Guttman,** USA
END OF FRIDAY Hall- CAJAL	

Friday April 05, 2019	**Hall- PICASSO**
07:30-08:30	**Chairpersons: Zaid Afawi,** Israel **& Elinor Ben-Menachem,** Sweden **FREE COMMUNICATIONS, EPILEPSY**
07:30-07:40	**Overlap of the Pitt–Hopkins and Lennox-Gastaut syndromes: Biljana Dapic Ivancic,** Croatia
07:40-07:50	**Prevalence of headache among patients with epilepsy: Ewa Czapińska-Ciepiela,** Poland
07:50-08:00	**Development of patients` e-registry and electronic medical records (EMR) as cost-effective management system for epilepsy - the pilot study in Georgia: Sofia Kasradze,** Georgia
08:00-08:10	**Parietal lobe, thermorgulation, and febrile seizures in an evolutionary quest: Alexandra Kunz,** USA
08:30-10:10	**SESSION 9 | IMMUNE THERAPY IN EPILEPSY; NON EPILEPTIC SEIZURES: PSYCHOGENIC OR NOT?**
Chairpersons:	**Olena Tsurkalenko,** Ukraine & **Nandan Yardi,** India
08:30-09:20	**Should we routinely prescribe immune modulatory therapy to patients with refractory adult-onset epilepsy who also develop psychiatric or cognitive impairment?** *Capsule: Autoimmune epilepsy is often accompanied by cognitive, behavioral, psychiatric or motor symptoms. However, such symptoms are often present in epilepsy patients without an autoimmune cause. Diagnosis of an autoimmune disease may be challenging.Should autoimmune treatment be initiated in people without known antibodies who have accompanying symptoms?*
08:30-08:40	Host: **Dana Ekstein,** Israel
08:40-08:55	Pro: **William Theodore,** USA
08:55-09:10	Con: **Martin Holtkamp,** Germany
09:10-09:20	Discussion and rebuttals
09:20-10:10	**Are non-epileptic seizures really psychogenic?** *Capsule: A variety of non-epileptic behaviors may be misdiagnosed as epileptic seizures. Many are deemed psychogenic in nature, particularly when co-existing psychiatric morbidity is present. Is the presumption of a psychogenic cause supported by evidence?*
09:20-09:30	Host: **Alla Guekht,** Russia
09:30-09:45	Yes: **Curt W LaFrance,** USA
09:45-10:00	No: **Amos Korczyn,** Israel
10:00-10:10	Discussion and rebuttals
10:10-10:25	***Coffee Break***
10:25-12:05	**SESSION 10 | TREATMENT OF RESISTANT SEIZURES**
Chairpersons:	**Nana Tatishvili,** Georgia **& Arie Weinstock**, USA

10:25-11:15	**Should antiepileptic drugs (AED) be pushed to high doses and levels before switching to or adding a new drug?** *Capsule: Traditional practice has been to raise doses of AED to achieve relatively high levels before switching to or adding another agent. Is this practice appropriate, or is failure at low dose indicative of treatment failure?*
10:25-10:35	Host: **Manuel Toledo,** Spain
10:35-10:50	Yes: **Elinor Ben-Menachem,** Sweden
10:50-11:05	No: **Martin Brodie,** UK
11:05-11:15	Discussion and rebuttals
11:15-12:05	**Should vagus nerve stimulation (VNS) be recommended early in the course of illness when seizures fail to respond to medication and cause falling or generalize?** *Capsule: VNS has the potential to moderately reduce seizure frequency. Should early use be advised primarily for patients whose seizures may cause injury, or should VNS be more broadly applied? What benefits would be expected in either situation – do patients with non-injurious seizures gain sufficiently to warrant treatment?*
11:15-11:25	Host: **Zeljka Petelin Gadze**, Croatia
11:25-11:40	Pro: **Antonio Gil-Nagel**, Spain
11:40-11:55	Con: **Ivan Rektor**, Czech Republic
11:55-12:05	Discussion and rebuttals
13:15-14:15	***Lunch Break***
13:15-:14:15	**Meet the Expert –Epilepsy** (Lafora) **Spotlight on the antiepileptic drug eslicarbazepine acetate: sharing experience from clinical practice.** **Vicente Villanueva,** Spain
14:15-15:45	**SESSION 11 | LACTATION IN EPILEPSY; CANNABIS?**
Chairpersons:	**Andry Dubenko,** Ukraine & **Xiana Rodríguez Osorio,** Spain
14:15-14:55	**Should women breastfeed if they take anticonvulsant medication?** *Capsule: Breastfeeding is generally recommended as a healthy practice. However, antiepileptic drugs are delivered to babies via breast milk. Is breastfeeding a sensible and safe practice for a baby whose mother takes an antiepileptic drug?*
14:15-14:25	Host: **Ilan Blatt,** Israel
14:25-14:35	Yes: **Martin Brodie,** UK
14:35-14:45	No: **Alla Guekht,** Russia
14:45-14:55	Discussion and rebuttals
14:55-15:45	**Should we prescribe medical marijuana for adult patients with drug-resistant epilepsy?** *Capsule: Some chemical constituents of marijuana may have anti-seizure effects, and Dravet and Lennox-Gastaut syndromes respond to cannabidiol. Do we know enough about medical marijuana to advise its use in adults with refractory epilepsy?*
14:55-15:05	Host: **Martin Holtkamp,** Germany
15:05-15:20	Yes: **Elson So,** USA

15:20-15:35	No: **Ilan Blatt,** Israel
15:35-15:45	Discussion and rebuttals
15:45-16:00	***Coffee Break***
16:00-19:00	**SESSION 12 | EPILEPSY: ADVANCED MRI; GENETICS**
Chairpersons:	**Juan José Poza**, Spain & **Tetyana Litovchenko,** Ukraine
16:00-16:50	**Are genetic data likely to be of major importance in the personalized treatment of epilepsy patients?** *Capsule: In addition to being causative in some rare epilepsies, genetic variants may play a role in susceptibility to more common types of epilepsy. Can these genetic features be used to guide management in individual patients?*
16:00-16:10	Host: **Michael Sperling,** USA
16:10-16:25	Likely: **Samuel Berkovic,** Australia
16:25-16:40	Unlikely: **William Theodore,** USA
16:40-16:50	Discussion and rebuttals
16:50-17:40	**Should MRI scans undergo routine post-processing if visual inspection does not show abnormalities in people with epilepsy?** *Capsule: A variety of sophisticated computer techniques can be employed in the analysis of MRI scans. When visual inspection fails to reveal an abnormality, do these techniques improve diagnosis, and is their use worthwhile?*
16:50-17:00	Host: **Manuel Toledo,** Spain
17:00-17:15	Yes: **Matthias Koepp,** UK
17:15-17:30	No: **Elson So,** USA
17:30-17:40	Discussion and rebuttals
17:40-19:00	**Epilepsy Cases, Michael Sperling,** USA, and **Manjari Tripathi**, India *Capsule: Challenging cases will be presented to participants for discussion*
END OF FRIDAY HALL- PICASSO	

Friday April 05, 2019	**Hall- DE FALLA**
07:30-08:30	**Stroke Free Communications** **Chairpersons: Ghassan Balousha,** Palestine **& Karl Matz,** Austria
07:30-07:40	**Prognostic value in functional outcome of risk factors for ischemic stroke including laterality: a cohort study: Jorge Celis,** Colombia
07:40-07:50	**Plasminogen enhances the process of angiogenesis after cerebral ischemia in mice via thrombospondin: Jinghuan Fang,** China
07:50-08:00	**Spinal cord infarction by thoracic vertebral hemangioma - a case report: Meri Papajani,** Albania
08:00-08:10	**Acute stroke care in a stroke center in Delhi: challenges and learnings: Sanjay Saxena,** India
08:30-10:10	**SESSION 13 | STROKE PREVENTION**

Chairpersons:	**Ghassan Balousha,** Palestine & **Exuperio Diez Tejedor,** Spain
08:30-09:20	**Is pollution a major contributor to acute stroke on a global scale?** *Capsule: Air pollution contributes to increased morbidity and mortality from pulmonary and circulatory disorders. The role of particulate exposure to the risk of stroke is not fully defined but may be important. Is there sufficient clinical evidence implicating pollution as a major modifiable risk factor for stroke and can it be reduced with preventative measures?*
08:30-08:40	Host: **Adrian Parry-Jones, UK**
08:40-08:55	Pro: **Karl Matz,** Austria
08:55-09:10	Con: **Vida Demarin**, Croatia
09:10-09:20	Discussions and Rebuttals
09:20-10:10	**Is the polypill a valid concept for prevention of stroke?** *Capsule: Most patients with stroke require treatment of multiple modifiable vascular risk factors. Does the development of a "polypill" that contains antithrombotic, antihypertensive and cholesterol-reducing drugs improve compliance to treatment and are such pills as effective as the individual drugs?*
09:20-09:30	Host: **Adrian Parry-Jones, UK**
09:30-09:45	Yes: **Karl Matz,** Austria
09:45-10:00	No: **Laszlo Csiba**, Hungary
10:00-10:10	Discussions and Rebuttals
10:10-10:25	***Coffee Break***
10:25-12:05	**SESSION 14 | ANTICOAGULATION IN STROKE**
Chairpersons:	**Vitalii Goldobin**, Russia **&** **Aleksandras Vilionskis,** Lithuania
10:25-11:15	**Is the demonstration of a high number of cerebral microbleeds (CMBs) a contraindication to anticoagulant treatment?** *Capsule: Intracerebral hemorrhage (ICH) occurs in patients receiving anticoagulation. This risk may be higher in patients in whom CMBs are identified on MRI. The best management of anticoagulant treatment in patients with high CMB score in not clear. How should patients with high-risk of embolic stroke in whom anticoagulation therapy is indicated but in whom MRI shows CMBs be managed?*
10:25-10:35	Host: **Laszlo Csiba**, Hungary
10:35-10:50	Yes: **David Werring**, UK
10:50-11:05	No: **Mahmut Edip Gurol**, USA
11:05-11:15	Discussions and Rebuttals
11:15-12:05	**What is the best prevention strategy following acute stroke for patients with embolic strokes of undetermined source (ESUS): direct acting oral anticoagulants (DOACs) or anti-platelet medications?** *Capsule: Two recent large trials with DOACs in patients with ESUS showed no superiority of DOACs over aspirin. Do the results from NAVIGATE-ESUS and RESPECT-ESUS suggest that there is no place for DOACs in ESUS patients? The debate will focus on whether patients with suspected cardiac embolic source should be treated long-term with DOACs to prevent further embolic events, or is treatment with antiplatelet drugs justified?*

11:15-11:25	Host: **George Chrysant,** USA
11:25-11:40	DOACs: **Georgios Tsivgoulis,** Greece
11:40-11:55	Antiplatelets: **Jonathan Streifler,** Israel
11:55-12:05	Discussions and Rebuttals
13:15-14:15	***Lunch Break***
14:15-15:45	**SESSION 15 | STROKE THERAPY**
Chairpersons:	**Maia Beridze,** Georgia **Antonio Davalos**, Spain
14:15-14:55	**Collateral enhancement: Is there sufficient evidence to offer to patients with acute stroke?** *Capsule: The speed with which irreversible injury develops following an acute stroke is variable. The presence of good pial collateral arteries is perhaps the most important factor associated with slow progression of injury following an acute stroke. But is there sufficient evidence that collateral enhancement can improve stroke outcome and can we apply such therapies in routine patient care?*
14:15-14:25	Host: **Natan Bornstein,** Israel
14:25-14:35	Yes: **Ashfaq Shuaib**, Canada
14:35-14:45	No: **Georgios Tsivgoulis**, Greece
14:45-14:55	Discussions and Rebuttals
14:55-15:45	**Is there sufficient evidence for closure of patent foramen ovale (PFO) in ALL patients after TIAs and acute stroke?** *Capsule: PFO is a frequent finding on echocardiography done as part of acute stroke investigation. However, not all strokes are necessarily due to its existence. Therefore, although recent studies have provided evidence that PFO closure is superior to medical therapy alone, it is debatable whether closure should be recommended to all patients with demonstrated PFO.*
14:55-15:05	Host: **George Chrysant,** USA
15:05-15:20	Yes: **Krassen Nedeltchev**, Switzerland
15:20-15:35	No: **Jonathan Streifler,** Israel
15:35-15:45	Discussions and Rebuttals
15:45-16:00	***Coffee Break***
16:00-19:00	**SESSION 16 | ENDOVASCULAR TREATMENT (EVT)**
Chairpersons:	**Zdravka Poljakovic,** Croatia **David Werring**, UK
16:00-16:50	**Acute stroke patients with suspected large vessel occlusion (LVO): Should they be transferred directly to a comprehensive stroke center (CSC) or for initial assessment at primary stroke center (PSC)?** *Capsule: EVT for acute ischemic stroke patients with LVO is a safe and effective treatment for selected patients up to 24 hours. For those arriving up to 4.5 hours from onset, IV tPA is still recommended. However, its impact is questionable. This can have a major impact on where we decide to transfer patients, first to the nearest PSC for IV tPA treatment and then to the CSC or directly to CSC.*
16:00-16:10	Host: **Antonio Davalos**, Spain
16:10-16:25	Direct: **Natalia Perez de la Ossa**, Spain
16:25-16:40	PSC first: **Roni Eichel**, Israel
16:40-16:50	Discussions and Rebuttals

16:50-17:40	**Should thrombectomy be performed on extremes (mild stroke or low infarct volume)?** *Capsule: EVT for acute ischemic stroke patients with LVO in the anterior circulation is safe and has been shown to be most effective when performed on patients with moderate and severe strokes. Little is known about the safety and efficacy of EVT in those patients with mild stroke (<5 NIHSS) or moderate to severe ischemic changes in the admission CT.*
16:50-17:00	Host: **Roni Eichel**, Israel
17:00-17:15	Pro: **Marc Ribo**, Spain
17:15-17:30	Con: **Ashfaq Shuaib,** Canada
17:30-17:40	Discussions and Rebuttals
17:40-18:30	**Should secondary stroke prevention include DOACs in addition to aspirin?** *Capsule: Despite the significant benefits of antiplatelet therapy, stroke victims remain at high risk of stroke recurrence. Long-term vitamin K antagonist therapy was superior to aspirin monotherapy but increased the risk of bleeding. Is combined therapy justified?*
17:40-17:50	Host: **Natan Bornstein,** Israel
17:50-18:05	Yes: **Laszlo Csiba,** Hungary
18:05-18:20	No: **Jonathan Streifler,** Israel
18:20-18:30	Discussions and rebuttals
18:30-19:00	**Intracerebral hemorrhage (ICH)-new frontiers**: **Mahmut Edip Gurol**, USA
END OF FRIDAY HALL- DE FALLA	

Friday April 05, 2019	**Hall-CERVANTES**
08:00-08:30	**ALZHEIMER'S DISEASE FREE COMMUNICATIONS** **Chairpersons: Nataliya Pryankova,** Ukraine & **Gabriel Vainstein,** Israel
08:00-08:10	**Use [18]f-fluoro- deoxyglucose positron emission tomography and other biomarkers to assess risk of clinical progression in patients with amnestic mild cognitive impairment**: **Maria Sagrario Manzano,** Spain
08:30-10:10	**SESSION 17 | ALZHEIMER'S DISEASE (AD)**
Chairpersons:	**Nataliya Pryankova,** Ukraine & **Gabriel Vainstein,** Israel
08:30-09:20	**Is the evidence sufficient to recommend dietary interventions to reduce the risk of AD progression?** *Capsule: Extensive epidemiologic evidence implicated modifiable metabolic and dietary factors in increasing the risk of dementia, including AD, and several interventions have shown promise in early trials. Definitive RCTs involving nutritional interventions to prevent the progression of cognitive decline in AD are eagerly awaited, but what do we need to do meanwhile?*
08:30-08:40	Host: **Yvonne Freund-Levi,** Sweden
08:40-08:55	Yes: **Aron Troen,** Israel
08:55-09:10	No: **Tobias Hartmann,** Germany
09:10-09:20	Discussions and Rebuttals

09:20-10:10	**Should cognitive disorders in older age be studied with FDG PET and amyloid PET or with MRI and CSF evaluation?** *Capsule: The clinical evaluation alone will misclassify about 20% of patients with dementia and a larger proportion of those with mild cognitive impairment. For this reason, biomarkers are used to help separate AD from frontotemporal dementia, which are treated differently. For this purpose, is it better to use PET metabolic biomarkers or MRI and CSF evaluation?*
09:20-09:30	Host: **Maria Sagrario Manzano,** Spain
09:30-09:45	Pro FDG and amyloid PET: **Joseph Masdeu**, USA
09:45-10:00	Pro MRI and CSF: **Guillermo Garcia Ribas,** Spain
10:00-10:10	Discussion and rebuttals
10:10-10:25	***Coffee Break***
10:25-12:05	**SESSION 18 | RISK FACTORS FOR AD**
Chairpersons:	**Shira Knafo,** Spain **& Mee Young Park**, South Korea
10:25-11:15	**There is no need to define dementia sub-types in older patients, as the majority have mixed pathologies anyway.** *Capsule: Researchers who examined older adults' brains after death found that most had two or more pathologies. Amyloid and tau were the most common pathology but rarely occurred alone. So, if the majority of older patients have mixed dementia, it may not be worthwhile to attempt to make a firm clinical diagnosis?*
10:25-10:35	Host: **Pierre Krolak-Salmon,** France
10:35-10:50	Pro: **Pasquale Calabrese,** Switzerland
10:50-11:05	Con: **Michael Ewers,** Germany
11:05-11:15	Discussion and rebuttals
11:15-12:05	**Microglia activation should be a therapeutic target.** *Capsule: Microglia activation and other innate immune responses seem to be associated with most neurodegenerative conditions, including AD. Is microglia activation merely a non-specific response to AD pathology or should it be considered a potential therapeutic target?*
11:15-11:25	Host: **Robert Perneczky**, Germany
11:25-11:40	Pro: **Roger Bullock**, UK
11:40-11:55	Con: **Giulio Maria Pasinetti,** USA
11:55-12:05	Discussion and rebuttals
13:15-14:15	***Lunch Break***
13:15-14:15	**Meet the Expert- Alzheimer's disease** (Rio Hortega) **AMBAR (Alzheimer´s Management By Albumin Replacement) Trial Results: Clinical and Biomarker Update** **Laura Núñez,** Spain **Javier Olazarán,** Spain
14:15-15:45	**SESSION 19 | MIXED DEMENTIA**

Chairpersons:	**Judith Aharon**, Israel **&** **Angel Martin Montes,** Spain
14:15-14:55	**Is APOE4 really toxic in AD?** *Capsule: The ε4 allele of apolipoprotein E (APOE) is the major genetic risk factor for AD. Many studies suggest that the differential effects of APOE isoforms on Aβ aggregation and clearance play the major role in AD pathogenesis. Inconsistent results among studies have made it difficult to define whether the APOE ε4 allele represents a gain of toxic function, a loss of neuroprotective function, or both.*
14:15-14:25	Host: **David Knopman,** USA
14:25-14:35	Pro: **Danny Michaelson**, Israel
14:35-14:45	Con: **Illiya Lefterov**, USA
14:45-14:55	Discussion and rebuttals
14:55-15:45	**Vascular risk factors in AD - real or fake?** *Capsule: Aging is associated with a large increase in the prevalence and incidence of degenerative and vascular dementia. Several vascular risk factors have been found to be associated with vascular dementia but also AD. Vascular risk factors and their treatments are a promising avenue of research for prevention of dementia, but do they really affect AD?*
14:55-15:05	Host: **Maria Sagrario Manzano,** Spain
15:05-15:20	Real: **Jan Kassubek**, Germany
15:20-15:35	Fake: **Giancarlo Logroscino**, Italy
15:35-15:45	Discussion and rebuttals
15:45-16:00	***Coffee Break***
16:00-17:40	**SESSION 20 | DEMENTIA CAUSES**
Chairpersons:	**Nina Sofilkanych**, Ukraine **&** **Ascensión Zea-Sevilla,** Spain
16:00-16:50	**The recent reduction of dementia incidence can be ascribed mainly to better management of hypertension, dyslipidemia and diabetes.** *Capsule: The prevalence of dementia is expected to soar as the average life expectancy increases, but recent epidemiological results suggest that the age-specific incidence of dementia is declining. We are going to discuss these results: is prevention possible?*
16:00-16:10	Host: **Michael Ewers,** Germany
16:10-16:25	Yes: **Milica G. Kramberger**, Slovenia
16:25-16:40	No: **Roger Bullock**, UK
16:40-16:50	Discussion and rebuttals
16:50-17:40	**Have we got it all wrong? Amyloid cascade is not the key etiological factor in AD.** *Capsule: The dominant hypothesis of AD etiology which has been built around one casual factor only, ß-amyloid (Aß), remains unproven. No conclusive evidence has been presented that Aß pathology represents the first biomarker of the disease and the first sign of sporadic AD onset. Treatments aiming to reduce Aß formation have proven to be toxic or worsen cognition. Immunization with anti Aß antibodies has not yet demonstrated a clinical effect. Should we discard the amyloid hypothesis?*
16:50-17:00	Host: **Ruth Itzhaki,** UK
17:00-17:15	Pro: **Ezio Giacobini**, Switzerland
17:15-17:30	Con:

17:30-17:40	Discussion and rebuttals
17:40-19:20	**SESSION 21 | AD: CAUSE AND THERAPY**
Chairpersons:	**Mun Seong Choi**, South Korea **& Latchezar Traykov**, Bulgaria
17:40-18:30	**Is herpes virus infection a risk factor for AD?** *Capsule: Herpes simplex virus type 1 (HSV1), when present in the brain of carriers of APOE4, has been implicated as a major factor in AD. It is proposed that virus is normally latent in many elderly brains but reactivates periodically. Implicating HSV1 further in AD is the discovery that HSV1 DNA is specifically localized in amyloid plaques in AD. Can we implicate HSV in AD pathogenesis?*
17:40-17:50	Host: **David Knopman,** USA
17:50-18:05	Yes: **Ruth Itzhaki,** UK
18:05-18:20	No: **Israel Steiner**, Israel
18:20-18:30	Discussion and rebuttals
18:30-19:20	**Is non-invasive brain stimulation (NIBS) useful for improvement of cognition in MCI subjects?** *Capsule: NIBS techniques include repetitive transcranial magnetic stimulation (rTMS) and transcranial direct current stimulation (tDCS). While these have been mostly used to treat pharmaco-resistant depression, mild cognitive impairment has also been reported to improve. However, the question remains: Is NIBS really useful for modulation of cognition in MCI?*
18:30-18:40	Host: **Jack de la Torre,** USA
18:40-18:55	Yes: **Irena Rektorova**, Czech Republic
18:55-19:10	No: **Friedhelm Hummel,** Switzerland
19:10-19:20	Discussion and rebuttals
END OF FRIDAY HALL- CERVANTES	

Saturday April 06, 2019	**Hall-CAJAL**
07:00-08:00	**E-Poster Presentations** (in exhibition hall) **PD FREE COMMUNICATIONS** **Chairperson: Abdelhamid Benazzouz**, France **& Pablo Martinez-Martin,** Spain
07:00-07:10	**A provocative test as a new approach to preclinical diagnostics of Parkinson's disease and to assessment of degradation of nigrostriatal dopaminergic system: Michael Ugryumov,** Russia
07:10-07:20	**Study of olfactory function in patients with Parkinson disease and healthy people: Denis Pokhabov,** Russia
07:20-07:30	**Atypical parkinsonian tauopathies – different diseases?: Piotr Alster,** Poland
07:30-07:40	**Subjective and objective motor function is associated with prodromal Parkinson's disease: a population based cohort study: Georgia Xiromerisiou,** Greece
08:00-10:30	**SESSION 22 | IMAGING; WEARABLE TECHNOLOGY; ORTHOSTATIC HYPOTENSION**
Chairpersons:	**Oleg Levin**, Russia **& Georgia Xiromerisiou,** Greece
08:00-08:50	**DAT imaging with SPECT or PET in parkinsonism: which one to choose?**

	Capsule: It is sometimes important to differentiate between idiopathic PD and another parkinsonian syndrome. The most reliable biomarker is through imaging, which one to use?
08:00-08:10	Host: **Javier Arbizu,** Spain
08:10-08:25	Pro SPECT: **Pierre Payoux**, France
08:25-08:40	Pro PET: **Andrea Varrone**, Sweden
08:40-08:50	Discussion and rebuttals
08:50-09:40	**Wearable technology devices will replace clinical PD motor assessments.** *Capsule: The current standard of PD management relies on patient histories and neurological examinations. However the infrequent nature of medical visits limits the ability to optimize care. With wearable technologies, neurologists can now collect longer durations of patient information and utilize these continuous objective measures to tailor management and do so with greater precision.*
08:50-09:00	Host: **Raul Martinez Fernandez,** Spain
09:00-09:15	Pro: **Fatta Nahab**, USA
09:15-09:30	Con: **Pablo Martinez-Martin,** Spain
09:30-09:40	Discussion and rebuttals
09:40-10:30	**Neurogenic orthostatic hypotension is a major cause of disability in PD**. *Capsule: Orthostatic hypotension commonly occurs in PD, either as part of the disease or caused by drugs. Is it clinically important?*
09:40-09:50	Host: **Stuart Isaacson,** USA
09:50-10:05	Pro: **David Goldstein,** USA
10:05-10:20	Con: **Nestor Galvez Jimenez,** USA
10:20-10:30	Discussion and rebuttals
10:30-10:45	***Coffee Break***
10:45-12:25	**SESSION 23 | PD: PSYCHOSIS AND MOTOR FLUCTUATIONS**
Chairpersons:	**Victoria Gryb,** Ukraine, & **Diego Santos Garcia,** Spain
10:45-11:35	**Treating PD psychosis early improves long-term outcomes.** *Capsule: Psychosis is commonly observed as a consequence of PD therapy. However the type of perceptual disturbance or thought content varies. The co-occurrence of depression, psychosis and dementia in patients with PD may indicate a more widespread pathological process affecting many neurotransmitter systems. Would early treatment of psychosis improve long-term outcomes?*
10:45-10:55	Host: **Nestor Galvez Jimenez,** USA
10:55-11:10	Pro: **Daniel Kremens,** USA
11:10-11:25	Con: **Jaime Kulisevsky Bojarski**, Spain
11:25-11:35	Discussion and rebuttals

11:35-12:25	**Gastrointestinal dysmotility is the major cause of motor fluctuations in PD.** *Capsule: Erratic gastric emptying is certainly one cause for fluctuations in advanced disease. However, dopaminergic neurons depletion and limited levodopa storage are the classical causes of fluctuations. Then should we treat brain or should we treat stomach and gut in PD?*
11:35-11:45	Host: **Bogdan Popescu**, Romania
11:45-12:00	Pro: **Stuart Isaacson,** USA
12:00-12:15	Con: **Esther Cubo**, Spain
12:15-12:25	Discussion and rebuttals
12:25-13:25	***Lunch Break***
12:25-13:25	**Meet the Expert:** (Rio Hortega) **Emerging perspectives regarding the use of on-demand therapies to treat OFF episodes in PD.** **Per Odin,** Sweden **Mark Lew,** USA **Daniel Kremens,** USA
12:25-13:25	**Meet the Expert- PDMD** (Lafora) **Neurogenic orthostatic hypotension (NOH): I. Pathogenisis; II. Clinical diagnosis; III. Distinguishing NOH from OFF symptoms; IV. Currrent approach to NOH treatment** **Fiona Gupta,** USA; **Laxman Bahroo,** USA; **Stuart Isaacson;** USA
13:25-15:05	**SESSION 24 | DYSKINESIAS**
Chairpersons:	**Pablo Mir Rivera**, Spain & **Angela Deutschlaender,** USA
13:25-14:15	**Medical treatment of dyskinesias is as effective as deep brain stimulation (DBS)** *Capsule: Dyskinesias affect a significant proportion of patients with PD, and is mostly observed after disease durations of several years. The presence of severe motor fluctuations and dyskinesias is one of the most important reasons for clinicians to recommend DBS. Can medical treatment achieve a reduction of dyskinesias which is comparable to DBS?*
13:25-13:35	Host: **Fiona Gupta,** USA
13:35-13:50	Pro: **Rajesh Pahwa,** USA
13:50-14:05	Con: **Sharon Hassin-Baer,** Israel
14:05-14:15	Discussion and rebuttals
14:15-15:05	**Tardive dyskinesia (TD) remains a common consequence of conventional antipsychotics.** *Capsule: TD represents involuntary movements affecting face, trunk or extremities, usually occurring after treatment with antipsychotics. The prevalence of TD in patients treated with conventional antipsychotic drugs ranges between 20-50%, and atypical antipsychotic drugs are thought to carry a lower risk of TD. These involuntary movements may disappear after discontinuation of the incriminated drug. How do we do the long-term management of the psychiatric patient that developed TD?*
14:15-14:25	Host: **Pedro J. Garcia Ruiz**, Spain
14:25-14:40	Pro: **Laxman Bahroo,** USA
14:40-14:55	Con: **Cristian Falup-Pecurariu**, Romania
14:55-15:05	Discussion and rebuttals
15:05-15:20	***Coffee Break***

15:20-17:50	**SESSION 25 | ADVANCED DOPAMINERGIC THERAPIES IN PD**
Chairpersons:	**Miguel Aguilar-Barberá,** Spain **& Vladimira Vuletic,** Croatia
15:20-16:10	**Off time will disappear with longer acting levodopa (LD) formulations.**
	Capsule: The so called "honeymoon" period of good response to LD in PD lasts 5-7 years. The mechanisms responsible for the loss of smooth response are complex and include gastric emptying as well as pharmacokinetic and pharmacodynamic factors. Could a better LD formulation solve the problem?
15:20-15:30	Host: **Laxman Bahroo,** USA
15:30-15:45	Pro: **Diego Santos Garcia,** Spain
15:45-16:00	Con: **Jaroslaw Slawek,** Poland
16:00-16:10	Discussion and rebuttals
16:10-17:00	**Subcutaneous apomorphine infusion should be used before other advanced therapies.**
	Capsule: Subcutaneous apomorphine infusion and advanced therapies of motor symptoms of PD intrajejunal levodopa infusions and DBS, each with distinct side effects. The individual PD symptoms profile should be assessed in order to choose an optimal treatment option. Should we use apomorphine infusions prior to recommending DBS surgery or intrajejunal levodopa infusions?
16:10-16:20	Host: **Stuart Isaacson,** USA
16:20-16:35	Pro: **Mark Lew,** USA
16:35-16:50	Con: **Per Odin,** Sweden
16:50-17:00	Discussion and rebuttals
17:00-17:50	**Development of non-dopaminergic therapies is a greater unmet need than dopaminergic treatments.**
	Capsule: PD patients suffer motor and non-motor symptoms. Most motor symptoms are dopamine-responsive. But some motor symptoms, such as tremor, as well as non-motor symptoms, may not respond and even worsen with dopaminergic medication. The question therefore arises whether development of non-dopaminergic therapies is a greater unmet need than dopaminergic treatments.
17:00-17:10	Host: **Fiona Gupta,** USA
17:10-17:25	Pro: **Abdelhamid Benazzouz**, France
17:25-17:40	Con: **Ilana Schlesinger**, Israel
17:40-17:50	Discussion and rebuttals
17:50-19:00	**What should be the main therapeutic target in Huntington's disease (HD)?**
	Capsule: HD is an incurable neurodegenerative disease affecting adults. While chorea is the best known feature, patients also suffer from cognitive decline and other motor features. Which should be the main target for therapeutic intervention?
17:50-18:00	Host: **Jaime Kulisevsky Bojarski**, Spain
18:00-18:15	Chorea: **Esther Cubo**, Spain
18:15-18:30	Bradykinesia and axial impairment: **Pedro J. Garcia Ruiz,** Spain
18:30-19:00	Discussion and rebuttals
18:00-19:00	**Meet the Experts-**(Lorente de Nó)
	OFF episodes in PD: GI dysmotility and emerging non-oral, on-demand therapies

	Laxman Bahroo, USA; **Stuart Isaacson,** USA; **Fiona Gupta**, USA, **Mark Lew,** USA
END OF SATURDAY HALL- CAJAL	

Saturday April 06, 2019	**Hall-PICASSO**
07:00-08:00	**E-Poster Presentations**
08:00-09:40	**SESSION 26 | HEADACHE THERAPY**
Chairpersons:	**George Chakhava**, Georgia **& Angel Guerrero,** Spain?
08:00-08:50	**Cognitive-behavioral therapy and biofeedback training are as effective as preventive medication in some patients.**
	Capsule: Medication and psychological intervention are often used in primary headache disorders. Can cognitive-behavioral therapy and biofeedback training replace preventive medication including CGRP blockers?
08:00-08:10	Host: **Robert Shapiro,** USA
08:10-08:25	Yes: **Steve Baskin,** USA
08:25-08:40	No: **Mark Braschinsky,** Estonia
08:40-08:50	Discussion and rebuttals
08:50-9:40	**Monoclonal antibodies to CGRP will become first line treatment not only for migraine but also for episodic cluster headache.**
	Capsule: CGRP plays a crucial role in migraine pathophysiology. Monoclonal antibodies to CGRP or its receptor are promising new therapies for the treatment of other types of headache as well.
08:50-09:00	Host: **Christian Lampl,** Austria
09:00-09:15	Yes: **Lars Edvinsson,** Sweden
09:15-09:30	No: **Jose Miguel Lainez.** Spain
09:30-09:40	Discussion and rebuttals
09:40-10:30	**SESSION 27 | NON-PHARMACOLOGICAL TREATMENT FOR HEADACHE**
Chairpersons:	**Elsa Parreira,** Portugal **& Maria Magdalena Wysocka-Bakowsa,** Poland
09:40-10:30	**Electrical stimulation will replace medications for the treatment of cluster headache.**
	Capsule: Neurostimulation is a rapidly growing field in headache disorders and provides an alternative therapeutic option particularly for cluster headache.
09:40-09:50	Host: **Jack Schim,** USA
09:50-10:05	Yes: **Licia Grazzi,** Italy
10:05-10:20	No: **Giorgio Lambru,** UK
10:20-10:30	Discussion and rebuttals
10:30-10:45	***Coffee Break***
10:45-12:25	**SESSION 28 | HEADACHE THERAPY**
Chairpersons:	**Elliot Gross,** USA **& Ruta Mameniskiene**, Lithuania
10:45-11:15	**Update on monoclonal antibody therapies and CGRP receptor antagonists in primary headache-**

	Messoud Ashina, Denmark
11:15-11:45	**Pipeline in headache treatment- Alan Rapoport,** USA
11:45-12:25	**FREE COMMUNICATIONS HEADACHE**
11:45-11:55	**Chronic headache - clinical evaluation of the chronic inflammatory state with acute inflammation: Maria Angels Carrera,** Spain
11:55-12:05	**Application of the cluster headache severity scale in a Korean cohort of cluster headache: Soo-Jin Cho**
12:05-12:15	**Can treatment of bruxism reduce migraine pain? Faik Ilik**
12:25-13:25	***Lunch Break***
13:25-15:05	**SESSION 29 | HEADACHE: CONCEPT AND MECHANISMS**
Chairpersons:	**Gabriela Mihăilescu,** Romania **& Krystyna Mitosek-Szewczyk,** Poland
13:25-15:05	**Migraine with aura and migraine without aura are the same disease.** ***Supported by Teva*** *Capsule: It is often debated whether migraine with aura and migraine without aura are etiologically distinct disorders. Do they share common pathophysiological pathways, such as cortical spreading depression, blood flow changes, and genotype?*
13:25-13:35	Host: **Dimos Mitsikostas,** Greece
13:35-13:50	Yes: **Isabel Pavao Martins,** Portugal
13:50-14:05	No: **Margarita Sanchez-del-Rio**, Spain
14:05-14:15	Discussion and rebuttals
14:15-15:05	**Does the blood brain barrier (BBB) open during a migraine attack?** *Capsule: Disruption of the BBB and inflammation are important contributors to the pathogenesis of neurological disorders. Although inflammation has been implicated in migraine pathogenesis, it is not known whether barrier integrity is compromised during attacks.*
14:15-14:25	Host: **Jose Miguel Lainez,** Spain
14:25-14:40	Yes: **Pablo Irimia Sieria,** Spain
14:40-14:55	No: **Messoud Ashina,** Denmark
14:55-15:05	Discussion and rebuttals
15:05-15:20	***Coffee Break***
15:20-17:00	**SESSION 30 | HEADACHE DIAGNOSIS**
Chairpersons:	**Mark Braschinsky,** Estonia **& Parisa Gazerani,** Denmark
15:20-16:10	**Computers can diagnose cluster headache better than the average doctor** *Capsule: Personalized medicine (patient and doctor in the same room) is rapidly being replaced by modern e-techniques and information technology tools*
15:20-15:30	Host: **Min Kyung Chu,** South Korea
15:30-15:45	Yes: **Robert Cowan,** USA
15:45-16:00	No: **Giorgio Lambru,** UK
16:00-16:10	Discussion and rebuttals

16:10-17:00	**Thunderclap headache: Do we need more than head CT and lumbar puncture?** *Capsule: Thunderclap headache is often but not exclusively caused by subarachnoid hemorrhage. CT and lumbar puncture are indicated when patients present with thunderclap headache, but do we need more than that?*
16:10-16:20	Host: **Robert Cowan,** USA
16:20-16:35	Yes: **Christian Lampl,** Austria
16:35-16:50	No: **Julio Pascual,** Spain
16:50-17:00	Discussion and rebuttals
17:00-19:00	**SESSION 31 | HEADACHE**
Chairpersons:	**Theodoros Constantinidis,** Greece **&** **Ermal Kurmaku**, Albania,
17:00-17:50	**Medical cannabis is effective in chronic headache** *Capsule: The use of medical cannabis in patients with chronic headache varies widely, with contradicting data regarding its efficacy in chronic cluster headache, chronic migraine and chronic tension type headache.*
17:00-17:10	Host: **Manjit Matharu,** UK
17:10-17:25	Yes: **Brian McGeeney,** USA
17:25-17:40	No: **Dimos Mitsikostas,** Greece
17:40-17:50	Discussion and rebuttals
17:50-18:05	**New Players (Not for CME)** **Reimagine Migraine** **Germán Latorre González,** Spain
18:05-19:00	**Placebo and nocebo in headaches: Dimos Mitsikostas,** Greece
END OF SATURDAY HALL- PICASSO	

Saturday April 06, 2019	**Hall- DE FALLA**
07:00-08:00	**E-Poster Presentations**
08:00-10:30	**SESSION 32 | PROGRESSIVE MYOCLONUS EPILEPSIES (PME)**
Chairpersons:	**Eva Andermann**, Canada **&** **Rimma Gamirova**, Russia
	Capsule: PME's are rare, but very challenging epilepsies to manage. The majority of cases can now be given a specific diagnosis, and new disorders have been recently described. Here we will discuss the diagnostic approach, insights from the new genetics, treatment with conventional anti-epileptic drugs and emerging precision therapies.
08:00-08:05	Welcome, introduction, learning objectives: **Jose Serratosa**, Spain
08:05-08:35	PMEs: Clinical diagnosis, new forms and epilepsies on the borderland: **Samuel Berkovic**, Australia
08:35-09:05	Emerging treatments for the treatment of PME: **Pasquale Striano,** Italy

09:05-09:35	Enzyme replacement therapy for CLN2: **Marina Trivisano,** Italy
09:35-10:05	Lafora disease: Neurobiology and new therapeutic strategies: **Jose Serratosa**, Spain
10:05-10:30	Management of MERRF patients including myoclonic epilepsy: **Josef Finsterer,** Austria
10:30-10:45	***Coffee Break***
10:45-12:25	**SESSION 33 | NEUROIMMUNOLOGY: MYASTHENIA GRAVIS (MG) AND APLA SYNDROME**
Chairpersons:	**Eduardo Gomez-Utrero,** Spain **& Vitalie Lisnic,** Moldova
10:45-11:35	**Treatment of refractory MG.** *Capsule: Although MG is an overall success story in neurologic therapeutics, about 10% of the patients remain symptomatic despite treatments. Recently, Eculizumab , a monoclonal antibody against complement C5, was approved for treating refractory MG. Is such a clinical benefit sufficient to justify its use in considering its excessive cost of $500,000 per year?*
10:45-10:55	Host: **Bruno Gran,** UK
10:55-11:10	Yes: **Renato Mantegazza,** Italy
11:10-11:25	No: **Vivian Drory,** Israel
11:25-11:35	Discussion and rebuttals
11:35-12:25	**Should immunotherapy be part of first line treatment in APLA syndrome?** *Capsule: The antiphospholipid syndrome (APS) is formally defined by the presence of high titers of antibodies together with thrombotic arterial and venous events. The mainstay of treatment in patients with neurological manifestations of APS is anticoagulation which rarely affects the levels of the circulating antibodies and has significant risks. Furthermore, many of the neurological manifestations of APS may be due to direct effects of circulating antibodies. It is therefore open to debate whether the treatment of APS should include antibody lowering therapies, as is well established in other humural mediated autoimmune diseases*
11:35-11:45	Host**: Abhijit Chaudhuri**, UK
11:45-12:00	Pro: **Joab Chapman,** Israel
12:00-12:15	Con:
12:15-12:25	Discussion and rebuttals
	Lunch Break
13:25-15:05	**SESSION 34 | LIMBIC ENCEPHALITIS: NEUROMYELITIS OPTICA (NMO)**
Chairpersons:	**Rina Aharoni,** Israel & **Anastasios Orologas,** Greece
13:25-14:15	**Immunosuppresive/immunomodulating treatment in autoimmune limbic encephalities - when to stop? Based on clinical status or based on lab data?** *Capsule: Antibodies to cell-surface neuronal molecules (eg. LGI1, NMDAR) are diagnostic and causative in forms of autoimmune encephalitis, yet many express doubts about the usefulness of antibody levels during management. Are laboratory assays geared to diagnosis, but not follow-up? Can accurate measurements can be helpful in patient management more than clinical state?*
13:25-13:35	Host: **Friedemann Paul,** Germany
13:35-13:50	Clinical state: **Jacek Losy,** Poland
13:50-14:05	Lab data: **Angela Vincent,** UK
14:05-14:15	Discussion and rebuttals

14:15-15:05	**The future of NMO treatment is immune tolerance, not immunosuppression.** *Capsule: Neuromyelitis optica spectrum disorders are relapsing autoimmune disorders that often cause severe disability due to severe attacks and are treated typically with immunosuppression with potential side effects. Is immune tolerance the way forward or is it just a distant fantasy?*
14:15-14:25	Host: **Anu Jacob,** UK
14:25-14:40	Pro: **Brian Weinshenker**, USA
14:40-14:55	Con: **Hans Peter Hartung**, Germany
14:55-15:05	Discussion and rebuttals
15:05-15:20	***Coffee Break***
15:20-19:00	**SESSION 35 | NMO: WHEN TO STOP TREATMENT**
Chairpersons:	**Angela Vincent,** UK
15:20-16:10	**Immune suppression treatments can be withheld in NMO patients who have prolonged stability.** *Capsule: NMO is a demyelinating disease of the central nervous system which is characterized by episodes of optic neuritis and transverse myelitis. The best treatment approach currently available is using immunosuppressive drugs. Unfortunately, not always immunotherapy is successful and has to be changed. However, many patients can be stabilized for a long time.*
15:20-15:30	Host: **Brian Weinshenker,** USA
15:30-15:45	Pro: **Hans Peter Hartung,** Germany
15:45-16:00	Con: **Andrzej Glabinski**, Poland
16:00-16:10	Discussion and rebuttals
16:10-17:00	**Should immunosuppression be used in pregnant patients with NMO?** *Capsule: Attacks of NMO continue at the same frequency throughout pregnancy and increase in frequency postpartum; they and other consequences of NMO may have devastating consequences to mother and fetus. Can immunosuppressive drugs be safely administered or continued throughout pregnancy?*
16:10-16:20	Host: **Oscar Fernandez,** Spain
16:20-16:35	No: **Abhijit Chaudhuri**, UK
16:35-16:50	Yes: **Brian Weinshenker,** USA
16:50-17:00	Discussion and rebuttals
17:00-17:15	**Objective markers for onset of transthyretin familial amyloid polyneuropathy in asymptomatic ser77tyr mutation carriers: Amir Dori,** Israel
17:15-17:25	**MS Oral free communications**
17:15-17:25	**Autonomic symptom burden can predict disease activity in early MS**: **Tin Pavičić,** Croatia
END OF SATURDAY HALL- DE FALLA	

07:00-08:00	**E-Poster Presentations**
08:00-10:30	**SESSION 36 | NEUROREHABILITATION AFTER STROKE**
Chairpersons:	**Mihail Gavrilic & Sadagat Huseyova,** Azerbaijan
08:00-08:30	**Advances in neurorehabilitation science: the role of biomarkers as prognostic factors. Dafin Muresanu,** Romania *Capsule: Stroke recovery biomarkers could be used to understand mechanism, or predict recovery or treatment response. This is beneficial for patients, caregivers and clinicians as well as for planning subsequent clinical pathways and goal setting.*
08:30-09:20	**Paving the way to successful neurorehabilitation after stroke: is thrombolysis enough?** *Capsule: Thrombolysis/thrombectomy are standard therapy for acute ischemic stroke but have limited effect. Can it be enhanced when employed in combination with multi-modal therapeutic agents?*
08:30-08:40	Host: **Dafin Muresanu,** Romania
08:40-08:55	No: **Michael Chopp**, USA
08:55-09:10	Yes: **Ovidiu Bajenaru,** Romania
09:10-09:20	Discussion and rebuttals
09:20-10:10	**What is the best strategy for cognitive rehabilitation after stroke?** *Capsule: Cognitive deficits after stroke may affect the performance of some daily activities. Which is the best strategy for cognitive rehabilitation after stroke? The use of eHealth and Web-based architectures to implement information and communication technology systems will be also presented.*
09:20-09:30	Host: **José León-Carrión,** Spain
09:30-09:45	Classical techniques based on patient-therapist direct interaction: **Jozef Opara,** Poland
09:45-10:00	E-health information and communication technology: **José M. Cogollor,** Spain
10:00-10:10	Discussion and Rebuttals
10:10-10:30	**Free communications Rehab**
10:10-10:20	**Effects of action observation training in gait speed of stroke patients: a case series: Jeanelle Louise Dumalag,** Philippines
10:20-10:30	**Perception of burden and psychological stress in parents of hearing impaired and intellectually challenged children in Punjab: Nazia Mumtaz**, Pakistan
10:30-10:45	**Coffee Break**
10:45-12:25	**SESSION 37 | NEUROREHABILITATION OF COGNITIVE FUNCTIONS**
Chairpersons:	**Antonio Oliviero,** Spain
10:45-11:35	**Should we prefer a personalized cognitive home-based rehabilitation therapy for the brain damaged, over the traditional hospital-based comprehensive integrative approach?** *Capsule: Shortage of qualified personnel, constant increase in health care expenses and a steady increase in surviving people with disabilities, push the authorities to find other rehabilitative therapies than the traditional hospital-based model, such as home-based rehabilitation.*
10:45-10:55	Host: **Dafin Muresanu,** Romania
10:55-11:10	Personalized: **José M. Cogollor,** Spain
11:10-11:25	Traditonal: **Avi Ohry,** Israel

11:25-11:35	Discussion and Rebuttals
11:35-12:25	**Spinal cord injury: immediate decompression surgery or comprehensive conservative approach?** *Capsule: Spinal cord injuries have a tremendous medical, social and economical impact on individuals, families and society. The most controversial issue is the surgical versus conservative treatment immediately after the trauma.*
11:35-11:45	Host: **Dafin Muresanu,** Romania
11:45-12:00	Pro Conservative: **Avi Ohry,** Israel
12:00-12:15	Pro Surgical: **Natacha Leon,** Spain
12:15-12:25	Discussion and Rebuttals
12:25-13:25	**Lunch Break**
13:25-15:05	**SESSION 38 | NEURODEGENERATIVE DISEASES**
Chairpersons:	**Andrzej Friedman,** Poland **& Fernando de castro or francisco Javier vitorica**
13:25-14:15	**Are corticobasal degeneration (CBD) and progressive supranuclear palsy (PSP) interchangeable terms?** *Capsule: CBD and PSP are both 4 repeat tauopathies with rather heterogenous clinical presentations. However distinct differences underpin the notion that CBD and PSP are different diseases. Are these two manifestations of a spectrum disorder? This may have implications for designing future disease-modifying therapies.*
13:25-13:35	Host: **Isidro Ferrer,** Spain
13:35-13:50	Yes: **Lea Grinberg**, USA/Brazil
13:50-14:05	No: **Tamas Revesz**, UK
14:05-14:15	Discussions and rebuttals
14:15-15:05	**Are microbiota reasonable targets in the therapy of neurodegenerative diseases?** *Capsule: The human microbiome consists of trillions of commensal microbes, including bacteria, fungi, and viruses, which naturally reside within the human body and have been documented to affect epigenetic mechanisims, metabolic activity, and immune function. Is there enough evidence to implicate the microbiome in neurodegenerative diseases?*
14:15-14:25	Host: **Ilana Schlesinger**, Israel
14:25-14:40	Yes: **Bogdan Popescu,** Romania
14:40-14:55	No: **Peter Jenner,** UK
14:55-15:05	Discussion and rebuttals
15:05-15:20	***Coffee Break***
15:20-18:00	**SESSION 39 | NEURODEGENERATIVE DISEASES**
Chairpersons:	**Angel chamaorro**
15:20-16:10	**Is suspected non-amyloid pathology (SNAP) a pre-clinical state of AD?** *Capsule: SNAP is identified through a biomarker definition as subjects with neurodegeneration (ND+) but no evidence of β-amyloidosis (Aβ-). This definition can be applied to all individuals including normal and mild cognitive impairment. SNAP has a different genetic profile and prognosis, and could represent a different pathway leading to dementia or it could be an earliest stage of AD.*
15:20-15:30	Host: **Eugen Tarnow,** USA

15:30-15:45	Yes: **Giancarlo Logroscino,** Italy
15:45-16:00	No: **Lea Grinberg,** USA/Brazil
16:00-16:10	Discussion and rebuttals
16:10-18:00	**Round table discussion: Glia are centrally involved in the pathogenic process of degenerative diseases and should be a therapeutic target**. Host: **Antonio Federico,** Italy and **Rafael Franco**, Spain Speakers: **Peter Jenner,** UK **, Roger Bullock,** UK**, Fernando de Castro**, Spain; **Lea Grinberg**, USA/Brazil
END OF SATURDAY HALL- CERVANTES	

Sunday April 07, 2019	**Hall- DE FALLA**
07:00-08:00	**E-Poster Presentations**
08:00-10:00	**SESSION 40 | PARKINSONS DISEASE (PD): COPPADIS MEETING**
Chairpersons:	**Juan Carlos Martínez Castrillo,** Spain & **Jaime Kulisevsky Bojarski**, Spain
	Capsule: Well-designed, prospective studies for identifying PD progression biomarkers are necessary. COPPADIS-2015 (Cohort of Patient´s with Parkinson's Disease in Spain, 2015) is an observational, descriptive, 5-year follow-up, nationwide study with more than 1,000 subjects participating that try to provide important knowledge about PD progression. Here, we show some interesting data about this ongoing project.
08:00-08:30	COPPADIS-2015. Justification, objective and general aspects of the project: **Diego Santos Garcia,** Spain
08:30-08:50	Non-motor symptoms in PD: frequency, types and correlated factors. **Lluis Planellas Gine,** Spain
08:50-09:10	Depression (BDI-II) in PD: prevalence, types, and variables. **Miquel Aguilar Barberá**, Spain
09:10-09:30	Impulse control disorders and compulsive behaviours in PD. **Silvia Jesús Maestre**, Spain
09:30-09:50	Factors affecting quality of life in patients with Parkinson´s disease: motor vs non-motor symptoms. **Pablo Martínez-Martín**, Spain
09:50-10:00	Conclusion and future directions: **Diego Santos Garcia,** Spain, **Juan Carlos Martínez Castrillo,** Spain & **Jaime Kulisevsky Bojarski**, Spain
10:00-10:15	***Coffee Break***
10:15-13:00	**SESSION 41 | PARKINSON'S DISEASE**
Chairpersons:	**Nestor Galvez Jimenez,** USA
10:15-11:05	**Is vascular parkinsonism (VaP) is a useful clinical entity?** *Capusle: The diagnosis of VaP is based on convergence of clinical parkinsonism with variable pyramidal and ataxic motor and non-motor signs, such as cognitive changes or bladder incontinence, that are corroborated by anatomic or imaging findings of cerebrovascular disease. Some experts disagree.*
10:15-10:25	Host**: Fatta Nahab,** USA
10:25-10:40	Yes: **Ivan Rektor,** Czech Republic
10:40-10:55	No: **Oleg Levin,** Russia

10:55-11:05	Discussion and rebuttals
11:05-13:00	**Round table discussion: What is 'advanced PD' and how to select the best advanced treatment (apomorphine vs duodopa vs DBS)?** Host: **Rajesh Pahwa,** USA Participants: **Pedro J. Garcia Ruiz**, Spain; **Sharon Hassin-Baer,** Israel; **Mónica M Kurtis,** Spain; **Juan Carlos Martinez Castrillo**, Spain**; Irena Rektorova**, Czech Republic; **Jaroslaw Slawek**, Poland
13:00-13:15	**CLOSING CEREMONY: Amos Korczyn**. Israel Invitation to CONy 2020 Poster Awards

Sunday April 07, 2019	**Hall-CERVANTES**
07:00-08:00	**E-Poster Presentations**
08:00-10:00	**SESSION 42 | AMYOTROPHIC LATERAL SCLEROSIS (ALS)**
Chairpersons:	**Nana Kvirkvelia**, Georgia, **Juan Francisco Vazquez-Costa,** Spain
08:00-08:50	**Is the incidence of ALS increasing?** *Capsule*: C*ompared to epidemiological studies, more recent population-based surveys provide higher incidence rates of ALS. Is the disease becoming more frequent or perhaps this finding is a reflection of a more accurate diagnostic ascertainment? The aging of the population may explain a true increase but the detection of the disease in older individuals previously diagnosed with other clinical conditions offers an alternative explanation.*
08:00-08:10	Host: **Giancarlo Logroscino,** Italy
08:10-08:25	Pro**: Mónica Povedano Panades,** Spain
08:25-08:40	Con: **Ettore Beghi,** Italy
08:40-08:50	Discussion and rebuttals
08:50-10:00	**Should we offer a genetic test to all ALS patients?** *Capsule: There is increasing evidence that ALS has a multifactorial origin with interaction between genetic and environmental factors. Genes implicated in the disease were discovered, are also involved in other diseases. This makes counseling a complicate issue. Is the present evidence sufficient for offering genetic testing to newly diagnosed patients?*
08:50-09:00	Host: **Albert Ludolph,** Germany
09:00-09:15	Yes: **Antonio Federico,** Italy
09:15-09:30	No: **Vivian Drory,** Israel
09:30-09:40	Discussion and rebuttals
10:00-10:15	***Coffee Break***
10:15-12:45	**SESSION 43- ALS AND FTD; CAUSES OF ALS**
Chairperson:	**Eduardo Gonzalez Toledo kabussek**

10:15-11:05	**Is fronto-temporal dementia a nosologic entity distinct from ALS?** *Capsule: The discovery of the C9orf72 gene supported a genetic basis of ALS by increasing the proportion of patients with genetic susceptibility. However, the same gene has been implicated in the occurrence of fronto-temporal dementia. Is this finding sufficient to conclude that ALS and FTD are different aspects of the same disease or, given the multiple disease mechanisms attributable to our genes; they still are separate nosographic entities?*
10:15-10:25	Host: **Daniel Drubach,** USA
10:25-10:40	Yes: **Eugen Tarnow,** USA
10:40-10:55	No: **Vivian Drory,** Israel
10:55-11:05	Discussion and rebuttals
11:05-11:55	**Is statistical significance sufficient for recommending the use of a drug for ALS patients?** *Capsule: ALS is still considered an untreatable neurodegenerative disease. There are only two drugs, Riluzole and Edaravove that showed a statistically significant but a clinically modest efficacy in ALS patients. The use of a drug with modest efficacy does not have a significant impact on the progression of this devastating disease and increases the risk: benefit ratio of treatment. However, in the absence of effective treatments, is an at-best modest efficacy sufficient to give hope to the patient?*
11:05-11:15	Host: **Philippe Couratier**, France
11:15-11:30	Yes: **Albert Ludolph**, Germany
11:30-11:45	No: **Orla Hardiman,** Ireland
11:45-11:55	Discussion and rebuttals
11:55-12:45	**Is heavy physical exercise a risk factor for ALS?** *Capsule: Several studies investigated the association between ALS and physical exercise with contrasting findings. Although the role of intensive physical exercise may be detrimental to motor neurons and occupations implying heavy physical activities have been thought to increase the risk of ALS, there are reports showing protective effects of physical activity on ALS as with other neurodegenerative diseases. On this basis, should heavy physical exercise be considered a risk factor or a protective factor for ALS?*
11:55-12:05	Host: **Ettore Beghi,** Italy
12:05-12:20	Pro: **Philippe Couratier**, France
12:20-12:35	Con: **Orla Hardiman,** Ireland
12:35-12:45	Discussion and rebuttals